

# DISEASES OF THE NOSE, THROAT AND EAR

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## PREFACE TO THE NINTH EDITION

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THE opportunity afforded by a revision of this work has been utilized by some rearrangement, partial rewriting, and amplification of portions of the text. Obsolete matter has been eliminated and much new material added. Some illustrations have been replaced and many new ones introduced.

A new chapter, "Headaches and Neuralgias of the Face and Head," has been added which the authors hope will give some guidance in clarifying these symptoms of diverse and many times baffling origin.

Rhinoplastic reconstruction, so ably advocated by Dr. Fomon and others, has been described and the steps illustrated. Modifications or portions of this procedure may be utilized by the rhinologist in many septal deviations, combined with a nasal deformity, in which the classical submucous resection of the septum alone might not give an adequate airway.

Dr. J. D. Kelly has revised his technic of arytenoidectomy for bilateral paralysis of the recurrent laryngeal nerves. Dr. Alfred Lewy has revised the chapters on "Physiology and Functional Tests of the Labyrinth" and "Inflammatory Diseases of the Labyrinth." Drs. Gabriel Tucker and C. L. Jackson have revised their chapters on "Peroral Endoscopy."

William Lincoln Ballenger, Late Professor and Head of the Department of Otology, Rhinology and Laryngology, School of Medicine, University of Illinois, Chicago, was the sole author of the first four editions of this work. In the fifth and subsequent editions, the revisions have been entirely undertaken by myself. In this Ninth Edition, I have been assisted by Dr. John J. Ballenger.

H. C. B.

CHICAGO, ILLINOIS

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# DISEASES OF THE NOSE, THROAT AND EAR

## PART I

### THE NOSE AND ACCESSORY SINUSES

#### CHAPTER I

#### THE CLINICAL ANATOMY AND PHYSIOLOGY OF THE NOSE AND ACCESSORY SINUSES

##### THE ANATOMY OF THE NOSE

**The External Nose.**—The more or less pointed tip of the nose is known as the apex. Extending superiorly and somewhat posteriorly is the dorsum leading to the root of the nose where the dorsum merges

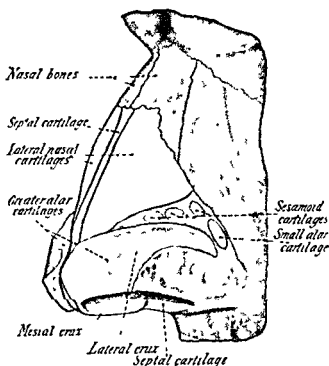


FIG 1 —The supporting framework of the external nose

with the forehead. The columella extends from the tip of the nose posteriorly and inferiorly to the lip. The point where the lip is reached is known as the base. On either side of the columella are the right and left anterior nares bounded laterally by the alæ of the nose and inferiorly by the floor.

The supporting framework of the external nose consists of the two nasal bones the frontal process (processus frontalis maxillæ) of the maxillary bones the paired upper and lower lateral cartilages and the unpaired anterior edge of the cartilaginous nasal septum. The cartilages on each side of the nose (Fig. 1) consist of an upper plate known as the upper lateral nasal cartilage a lower plate known as the lower lateral cartilage (greater alar cartilage) and one or more sesamoid cartilages placed between the two larger cartilages. The median portion (crux mediale) of the lower lateral cartilage extends along the free edge of the anterior inferior portion of the cartilaginous septum within the columella and participates in the formation of the anterior nares. The cartilages are closely attached to each other and to the nasal bones by strong connective tissue fibers.

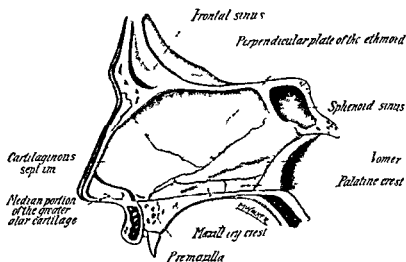


FIG. 2 The nasal septum denuded of its membrane

In the bony skull the pear shaped nasal opening is called the pyriform aperture. The superior lateral margins are formed by the nasal bones and frontal processes of the maxilla. The base is formed by the alveolar process of the maxilla. In the midline of this last structure is a prominence called the anterior nasal spine.

The alar muscles consist of two sets the dilators comprising the dilatores naris (anterior and posterior) the m. procerus and the caput angulare of the quadratus labii superioris and the constrictors comprising the m. nasalis the depressor septi and the depressor alæ nasi.

**The Nasal Septum**—The septum divides the nose into two cavities or chambers the right and the left. The septum (Fig. 2) is formed superiorly by the perpendicular plate of the ethmoid anteriorly by the septal (quadrilateral) cartilage premaxilla membranous columella and inferiorly and posteriorly by the vomer the maxillary crest the palatine crest and the sphenoidal crest.

**The Nasal Chambers — The Floor** — The floor of the nose is formed by the palatal process of the maxilla and the horizontal process of the palate bones.

**The Roof** — The roof from before backward is composed of the nasal bone, the nasal process of the frontal, the body of the ethmoid, and the body of the sphenoid. The lamina cribrosa or the cribriform plate of the ethmoid which forms the major portion of the roof of the nose transmits the filaments of the olfactory nerve as it descends from the under surface of the olfactory bulb to its distribution in the mucous membrane covering the upper portion of the superior turbinate and a corresponding portion of the septum.

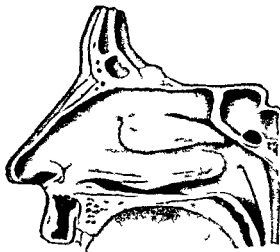


FIG. 3. The middle and inferior turbinates. The superior turbinate which may be absent or rudimentary is not shown.

**The Lateral Wall** — The lateral wall is formed by the inner surface of the frontal process of the maxilla, the lacrimal, the superior and middle nasal turbinates of the ethmoid, the inferior nasal turbinate, the perpendicular plate of the palate bone, and the medial pterygoid plate.

**The Turbinates (Conchæ)** — The nasal fossa is divided into three meati by the three turbinates. The space situated between the inferior turbinate and the floor is called the inferior meatus. The space between the middle turbinate and the inferior turbinate is known as the middle meatus, and above the middle turbinate is the superior meatus. Occasionally a fourth turbinate (supreme turbinate) is observed. The supreme, superior and middle turbinates originate from the lateral mass of the ethmoid. The inferior turbinate, a separate bone, is attached to the superior maxilla and to the palate.

The inferior turbinates are two elongated shell-like laminae of bone attached by their superior borders to the lateral wall of the nasal cavity on either side. They have curved borders separating a medial and a lateral surface. The inferior, or free portion is curved from before backwards and from above downward with the convex surface facing

the septum. The bone which forms the turbinate is deeply pitted and of somewhat cellular character which gives a slightly rough and pitted appearance. The anterior and posterior extremities are somewhat pointed. The surface of the turbinate is perforated in numerous places by apertures through which the blood supply is transmitted. Longitudinal grooves or partial canals also help distribute the large blood supply. The mucous membrane is thick, very vascular and adherent to the underlying perichondrium or periosteum.

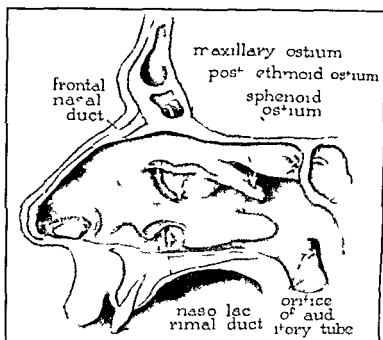


FIG. 4.—Sagittal section through the nose. Portions of the middle and inferior turbinates have been removed to show various structures.

Both the middle and inferior turbinates are covered with pseudo-stratified ciliated columnar epithelium and the anterior tip of either the middle or the inferior turbinates in the adult may be replaced by low cuboidal or squamous-cell epithelium. The stroma of the middle turbinate is characterized by the presence of many glands whereas that of the inferior turbinate is characterized by many blood lakes. Glands too are found in the inferior concha but not to the extent of the middle. These blood lakes or venous plexuses constitute the erectile tissue of the nose and are distributed chiefly along the inferior border of the inferior turbinate and the posterior ends of both the middle and inferior turbinates.

**The Superior Meatus**—The superior meatus or ethmoid fissure is a narrow slit like space situated between the septum and the lateral mass of the ethmoid. The posterior group of ethmoid cells drain by

one or more orifices of variable size into the central portion of the meatus. Above and behind the superior turbinate and in front of the body of the sphenoid is the sphenoid-ethmoidal recess, into which opens the sphenoid sinus.

**The Middle Meatus.**—The middle meatus, a much more roomy space than the superior meatus, contains the orifices of the frontal and maxil-

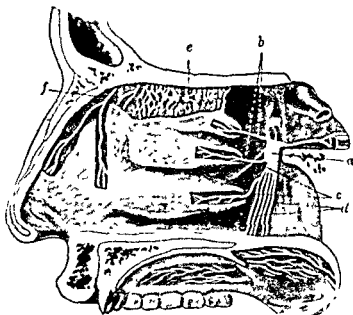


FIG 5 — Nerves of the lateral wall of the nose. *a*, sphenopalatine ganglion; *b*, posterior-superior lateral nasal branches; *c*, posterior-inferior lateral nasal branches; *d*, palatine nerve; *e*, olfactory nerve; *f*, internal nasal branch of the anterior ethmoid nerve (after Spalteholz).

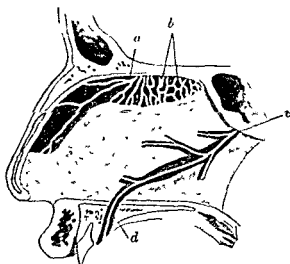


FIG 6 — Nerve supply of the septum. *a*, anterior ethmoid nerve; *b*, olfactory nerve; *c*, nasopalatine nerve; *d*, incisor canal (after Spalteholz).

lary sinuses and of the anterior group of ethmoid cells. Hidden by the anterior half of the overhanging middle turbinate and situated on the external wall is a deep crescentic groove, the *infundibulum*. The crescentic opening or fissure leading from the middle meatus into the *infundibulum* is called the *hiatus semilunaris*. The inferior and median wall of the *infundibulum* forms a shelf-like ledge known as the *uncinate process*. Above the *infundibulum* is a hemispherical prominence, the *ethmoid bulla*, formed by one of the ethmoid cells.

The orifices of the frontal sinus, maxillary antrum and anterior ethmoid cells usually drain into the *infundibulum*. The frontal sinus and anterior ethmoidal cells usually drain into the anterior upper portion, and the maxillary sinus drains posteriorly to the frontal sinus. However, certain ethmoid cells may have openings located above the *ethmoid bulla*, and the fronto-nasal duct sometimes has an independent orifice anterior to the *infundibulum*.

**The Inferior Meatus.**—The inferior meatus, the largest of the three, contains the orifice of the naso-lacrimal duct located on the lateral wall from 3 to 3.5 cm. behind the posterior margin of the nostril.

The posterior nares or choanae through which the nasal fossae and the nasopharynx communicate are oval-shaped apertures located one on each side of the nasal septum. Each opening is formed inferiorly by the horizontal plate of the palate bone, internally by the vomer, superiorly by the vaginal process of the sphenoid and the ala of the vomer, and externally by the medial pterygoid plate of the sphenoid.

### THE NASAL ACCESSORY SINUSES

The nasal sinuses are eight in number, four on each side of the nose: the right and left frontal sinuses, the right and left ethmoids (anterior and posterior), the right and left maxillary sinuses (antra of Highmore) and the right and left sphenoid sinuses. The sinuses are lined with the nasal mucous membrane, all are filled with air and all communicate with the nasal fossa through their various ostia.

The sinuses are divided for clinical purposes into two groups, namely, the anterior and the posterior sinuses. The anterior group is composed of the frontal, the anterior ethmoid, and the maxillary sinuses. Hajek calls this group Series I. The posterior group is composed of the posterior ethmoid and the sphenoid sinuses, and is called Series II.

**The Frontal Sinus**—The frontal sinus varies greatly in size and form and in many instances the sinus differs in extent and shape from its fellow, one sinus appearing to develop at the expense of the other. Occasionally the sinus is rudimentary but never entirely wanting. The sinus is not present at birth, first appearing about the third year and attaining its full size after puberty. At the seventh year it is about the size of a pea and consequently is of clinical importance from this age on. Bony septa may partially subdivide the sinus into one or more compartments. The sinus communicates with the middle meatus of the nose by means of the fronto-nasal duct, which passes downward and

backward and opens into or near the upper portion of the infundibulum. The fronto-nasal canal opens directly into the middle meatus in some instances.

The average measurements of the frontal sinus are, height 3 cm., width 2 to 2.5 cm., depth 1.5 to 2 cm., and average capacity 6 to 7 cc.

The anterior plate of the frontal sinus is almost always diploetic, especially in the regions of the external or infero-lateral angle and the superior sulcus where the anterior and posterior plates fuse.

**The Ethmoid Cells** The ethmoid cells or labyrinth lie on either side just lateral to the superior one-half or one-third of the nasal cavity and

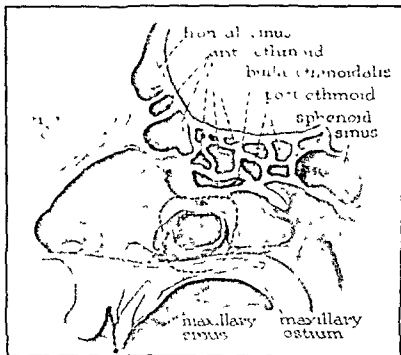


FIG. 7.—Sagittal section through the ethmoid labyrinth.

medial to the bony orbit. The ethmoid bone has a horizontal and a vertical plate which are at right angles to each other. The vertical plate has a superior thicker portion called the *crista galli* and an inferior portion called the *perpendicular plate of the ethmoid*, a part of the nasal septum.

The horizontal plate is comprised of a medial portion, the thin perforated *cribriform plate* and a more lateral, thicker portion which forms the roof of the ethmoid cells.

The cribriform plate is not covered by the cells, but is freely exposed in the attic of the nose. While the bone is dense and not easily fractured by ordinary force exerted during an operation, its numerous openings render it a possible atrium for the conveyance of infection to the meninges, especially if the ethmoid is operated upon in the presence



of an acute nose or throat infection. The outer wall of the ethmoid sinus is the *os planum* or *lamina papyracea* of the ethmoid and the lacrimal bones. These plates of bone are extremely thin and form the inner wall of the orbital cavity. Should this plate of bone be perforated orbital cellulitis with protrusion of the eyeball might result.

The ethmoid cells are present in the new born developing in size with advancing years until puberty. The ethmoid cells are a series of pneumatic cells of variable size and number situated in the upper part of the nose between the orbit and the outer wall of the nasal fossa. Two groups of cells may be differentiated. An anterior group which drain into the middle meatus and a posterior group which drain into the superior meatus.

The anterior cells are separated from the posterior cells by a thin transverse bony partition. The attachment of the middle turbinate to the external wall of the nose also marks the line of division between the anterior and the posterior group of cells. The anterior cells lie in front of and below it while the posterior cells lie above and behind it.

The posterior ethmoid cells are usually fewer in number and larger in size than the anterior ethmoid cells. Sometimes they occupy nearly all the ethmoid labyrinth extending to the anterior portion of the nose and sometimes the anterior cells extend backward almost to the sphenoid bone.

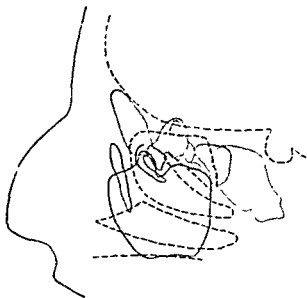
Frequently one or more ethmoid cells are found in the middle turbinate (concha bullosa). The ethmoid bulla is formed by an ethmoid cell usually of the anterior group. Frequently a large ethmoid cell projects into the frontal sinus producing what is known as a frontal bulla or frontal cell. The encroaching ethmoid cells may extend into the supra-orbital plate of the frontal bone forming supra-orbital cells.

The first sign of the future ethmoid cells is seen in the fourth month of fetal life when an outpouching of the nasal mucous membrane occurs. By birth the sinus is a fairly definite clinical entity.

**The Maxillary Sinus** — At birth the maxillary antrum occupies a small space to the inner side of the orbit. At first its floor is above the nasal floor descending continually until at eight years it is on the same level. The subsequent development is downward assuming its full shape after the eruption of the permanent teeth. The maximum development is attained between the fifteenth and eighteenth years. The maxillary sinus or the antrum of Highmore the largest of the nasal accessory sinuses is an irregularly shaped pyramid with its base presenting to the nasal fossa and its apex corresponding to the zygomatic process of the maxilla. It has a capacity of approximately 15 cc.

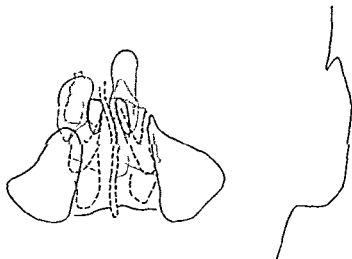
The median wall or base of the antrum is formed by the vertical plate of the palate bone the uncinate process of the ethmoid the maxillary process of the inferior turbinate and a small portion of the lacrimal bone. The upper wall separates the cavity from the orbit. The posterior inferior wall or floor is normally the thickest and is formed by the alveolar portion of the superior maxilla and by the outer part of the hard palate. The anterior wall corresponds with the canine fossa.

## PLATE I



Left Lateral Reconstruction (H W Loeb)

In these reconstructions the frontal sinuses are colored yellow, the maxillaries gray, the sphenoids green, and the ethmoids red, the anterior groups are lined horizontally and the posterior groups perpendicularly. In the lateral presentations the ethmoidal cells are in two groups, the anterior two in number, and the posterior three. The first anterior cell is shown displacing the anterior wall of the frontal. The frontal is seen opening into the frontonasal canal. The antero-inferior wall of the second ethmoid constitutes the bulla ethmoidalis.



Anterior Reconstruction (H W Loeb)

On account of the multiplicity of lines the individual ethmoidal cells are not shown, however the two groups are represented, the anterior being lined horizontally and the posterior perpendicularly. The left sphenoidal sinus lies far above the right, its inner wall extends almost as far to the right as the outer wall of the right sphenoidal sinus.

The antrum communicates with the infundibulum in the middle meatus by means of a small opening the maxillary ostium located in the upper and anterior part of the median sinus wall. In a small per-

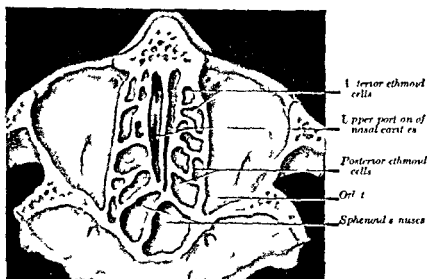


FIG. 8—Horizontal section through the ethmoid labyrinth

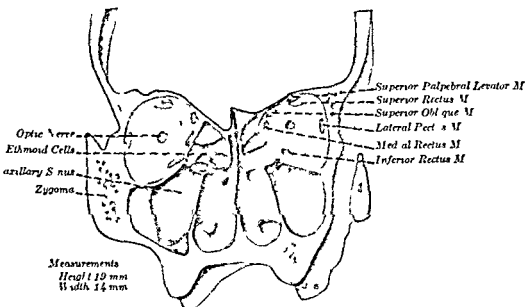


FIG. 9—Coronal section through the ethmoid labyrinth

centage (10 per cent) of cases in addition an opening (accessory ostium) is present lying posterior to the major opening. In the majority of cases the ostium is a canal of 3 mm or more in length. The accessory ostium is in almost all instances an orifice or true ostium. Most nerves,

and blood vessels enter the sinus by way of the ostium or the membranous portion of the infra-orbital wall.

The second bicuspid and the first and second molar teeth are in close relation to the floor of the sinus. Indeed they sometimes project into the bony cavity being covered at times by mucous membrane only. A suppurative process around the root of either of these teeth might

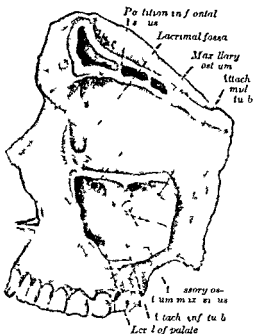


FIG. 10 — Medial antral wall showing the relationship of the intranasal structures.

affect the mucous membrane of the sinus through the lymphatics and blood vessels and the removal of these teeth may create an opening into the sinus with a resultant sinusitis.

The superior wall or roof of the sinus is crossed in its central portion by the infraorbital nerve which lies in a groove on the broad inferior side of the plate of bone. The nerve may be covered by mucous membrane or by thin bone and may be injured during the curettage of the sinus.

**The Sphenoid Sinus** — The sphenoid sinuses attain but small size before the third year, but are fully developed by the twelfth to the fifteenth year. They are situated within the body of the sphenoid bone, are variable in size and often in shape. They are separated from each other by a thin bony partition or septum which frequently deviates to one side or the other, producing one large and one small cavity.

Each sphenoid sinus communicates with the superior meatus of the nose by means of a small aperture which empties into the sphenoid recess. The size of the ostium of the sphenoid varies from pin

point size to almost complete absence of the nasal surface of the anterior wall. The ostium is practically always membranous its bony circumference being considerably larger than its actual orifice. It is near the septum of the nose and is hidden from view by the close approximation of the middle turbinate to the septum. If there is marked atrophy of the turbinate or if the septum deviates to the opposite side it may be seen by anterior rhinoscopy. The opening varies from  $\frac{1}{2}$  to 4 mm in diameter.

The purulent secretion flowing from the ostium either drains directly through the posterior choana into the nasopharynx or on to the posterior end of the middle turbinate. Direct inspection can be made as a rule only after the removal of the middle turbinate.

The superior wall of the sinus lies in relation to the cranial cavity. There is a close clinical relation of the sphenoid sinus to the third fourth fifth sixth and vidian nerves.

**The Development of the Sinuses**—The primordia (anlagen) of the sinuses originate rather late during the prenatal period especially that of the frontal sinus. During the first and second months of embryonic life the main features of the nasal cavities are differentiated. The sinuses arise as localized epithelial sprouts or recesses of the nasal mucosa after the second month. The recesses later become the ostia of the various sinuses.

The maxillary and sphenoid sinuses arise as mucosal recesses during the third prenatal month. At this time glandular sprouts develop from the mucosal recess in the hiatus semilunaris of the middle meatus to form the future maxillary sinus. At birth the maxillary sinus is a fairly well-developed tubular sac with its floor slightly below the superior border of the inferior meatus. After the seventh year the expansion of the maxillary sinus to its adult size and shape is relatively fast.

The sphenoid sinuses originate during the third fetal month as paired evaginations of the mucosa in the superior posterior portion of the nasal cavity. The development of the mucosal evaginations is very slow. Even at birth the mucosal cavities are not in relation to the posterior nasal cartilage or bony sphenoid (Zimmermann<sup>1</sup>). Pneumatization of the sphenoid bone occurs during middle childhood and proceeds rapidly after seven years of age to its final form and extent which is usually attained between twelve and fifteen years of age or even earlier.

The ethmoid cells originate during the fifth and sixth fetal months from the superior and supreme meatuses to form the anterior posterior and postreme groups of cells. The anterior group of cells derived from the middle meatus lies anterior as a rule to those cells originating from the superior meatuses. These cell groups expand unequally with great individual and group variations. The various groups of cells are fairly well formed at birth. The rounded epithelial recesses forming the cells are separated from each other by interspaces and bony septa. Growth of the cells is relatively rapid especially during the second year of life.

<sup>1</sup> Ann. Otol. Rhinol. and Laryngol. 47: 1112 (December) 1938.

At seven years of age most or all of the available space is pneumatized. Between twelve and fourteen years of age the cells have attained the final form.

Pneumatization of the frontal bone begins at the end of the first year of life by one of three ways: (1) by an expansion of the frontal recess in the upper anterior portion of the infundibulum; (2) by development of one of the frontal cells; and (3) by growth and expansion of a bulla cell. The location of the adult frontal ostium would vary somewhat depending upon the origin of the frontal sinus. The growth of the frontal sinus is slow up to the seventh year of age (size of a pea) and does not attain its adult size and form until from fifteen to twenty years of age.

THE DEVELOPMENT OF THE SINUSES FROM BIRTH

	At birth	1 yr	1 3 yrs	~ yrs	1 1/2 15 yrs	1 1/2 18 yrs
Maxillary	Size of small bean	Gradual development to				Fully developed
Ethmoid	Present	Gradual development to				Fully developed
Frontal	Absent	Absent	Appearance	Pea-size	Distinct cavity	Well developed
Sphenoid	Present as a rule	Distinct cavity	Size of large pea		Fully developed	

**The Nasal Mucous Membrane** — The nasal fossa and the sinuses are lined by mucous membrane of the columnar type continuous with that of the nasopharynx. The membrane is divided into two regions: the olfactory and the respiratory.

**The Olfactory Region** — The olfactory region is limited largely to the area occupied by the superior turbinates and the corresponding portions of the septum. The epithelium is non-ciliated columnar consisting of two chief types of cells: the supporting and the olfactory. Numerous serous glands (glands of Bowman) of a simple tubular character are present. The olfactory mucosa consists of a surface neuro-epithelium with a subjacent tunica propria. A definite and distinct basal membrane is wanting.

**The Respiratory Region** — The respiratory region embraces the remaining portion of the nasal mucous membrane. Portions of the mucosa are thick and very vascular as over the inferior turbinates where cavernous tissue is present. The mucous membrane is very adherent to the underlying periosteum or perichondrium and is of the pseudostratified ciliated columnar (respiratory) type except for the anterior ends of the middle and inferior turbinates and the mucous membrane of the lateral and septal walls anterior to these structures where non-ciliated stratified squamous epithelium is found. The respiratory mucous membrane rests upon a cribriform basement membrane. The ciliated cells extend through the entire thickness of the epithelium. The movement of the cilia is toward the posterior nares.

The glands of the mucous membrane are very numerous tubulo-alveolar in form and consist of mucous, albuminous and mixed types.

The albuminous glands secrete a thin serous secretion and the mucous glands the heavier, thicker mucus which together make up the moving blanket of mucus so important to the physiology of the nose.

**The Mucous Membrane Lining the Sinuses**—The paranasal sinus membrane is continuous with that of the nasal fossa. It is much thinner and has fewer glands except near the ostia of the maxillary and sphenoid sinuses.

The mucosa of the sinuses (Fig. 110) is composed of a pseudostratified ciliated columnar epithelium resting on a thin basal membrane and tunica propria somewhat adherent to the underlying periosteum. The movement of the cilia in the sinuses is toward their respective ostia.

**The Nerve Supply of the Nose**—**The Sensory Nerves**—The sensory nerves of the nasal mucous membrane are: (1) the anterior and posterior ethmoid branches of the ophthalmic branch of the trigeminus and (2) the branches of the sphenopalatine ganglion. The first branch of the trigeminal nerve, the ophthalmic, gives rise to the nasociliary nerve of which the chief terminal branches are the anterior and posterior ethmoid and the infratrochlear nerves. The anterior ethmoid nerve passes over the anterior portion of the cribriform plate (Fig. 6) through the anterior ethmoid foramen and thence forward and downward on the septum to supply the anterior margin of the septum and a portion of the lateral wall anterior to the turbinates including the anterior ends of the turbinates. The infratrochlear nerve is sensory to the external surface. The small and unimportant posterior ethmoid nerve passes over the cribriform plate and enters the nose through the foramen of the same name to supply a small area near its entrance point into the nose.

The *sphenopalatine (Meckel's) ganglion* lies deeply within the pterygopalatine fossa on either side just lateral to the sphenopalatine foramen. Its nasal (internal) branches are derived partly from the ganglion itself but consist largely of sensory fibers from the maxillary division of the trigeminal nerve which pass through the ganglion. These nasal branches pass through the sphenopalatine foramen as the sphenopalatine nerve and are distributed to the nasal septum (medial branches) and the lateral nasal wall (lateral branches). Some of the medial branches terminate in the posterior superior part of the septum but others are joined together to form the nasopalatine nerve (nerve of Cotunnus) which courses forward and downward to the incisive (anterior palatine) canal where it communicates with the anterior superior alveolar nerve. The lateral branches supply the mucous membrane of the middle and superior turbinates.

Other branches from the sphenopalatine nerve descend in the pterygopalatine canal to emerge at the greater palatine foramen on the under side of the hard palate. During its passage through the canal, branches are given off to the inferior turbinate. The terminal branches are distributed to the hard and soft palate, the uvula and tonsil.

Vasomotor branches from the sphenopalatine ganglion are also supplied to the vessels of the mucous membrane and erectile tissue of the turbinates and are under the control of the vasomotor center of the

medulla there is probably a connection with the nuclei of the vagus through association fibers

The sensory nerve supply of the external (skin) surface of the nose comes principally from three sources the infratrochlear nerve from the nasociliary the external nasal nerve which is one of the terminal branches of the anterior ethmoid nerve and the infraorbital nerve from the second division of the trigeminal

The *infratrochlear nerve* is distributed to the eyelids and medial portion of the eye and to the skin of the upper part of the side of the nose

The *external nasal nerve* after its origin from the anterior ethmoid runs downward in a groove on the inner surface of the nasal bone. It pierces the wall of the nose between the nasal bone and the upper lateral cartilage and supplies the skin of the lower part of the dorsum as far as the tip of the nose

The *infraorbital nerve* emerges on the cheek below the eye at the infraorbital foramen to supply a portion of the lateral wall of the nose and other structures of the face

Blocking of these nerves would produce anesthesia of the external portion of the nose

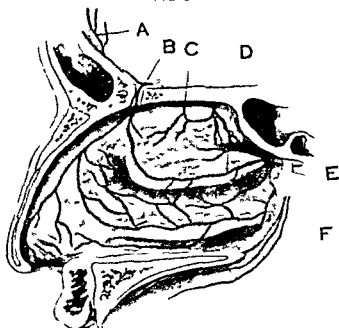
**The Olfactory Nerve**—The olfactory nerve descends through the cribriform plate (lamina cribrosa) from the under surface of the olfactory bulb and is distributed in the mucous membrane covering the upper portion of the superior turbinate and a corresponding portion of the septum. Formerly it was thought that the distribution of the olfactory nerve in man covered a much more extensive area the upper and median surfaces of the middle turbinate and a corresponding area of the septum being included in the alleged area of distribution. In many of the lower animals the nerve has a wider distribution the sinuses communicate more freely with the nasal chambers and are utilized for the spread of the terminal olfactory nerve filaments. In man they may be the remains of the organ of smell and only communicate with the nasal cavities through small ostia or cell openings as they are no longer needed for olfaction. It is obvious that if the middle turbinate and the septum are in apposition the inspired air does not reach the olfactory region and anosmia or loss of the sense of smell results

The *terminal nerve* originating from the terminal ganglion medial to the olfactory bulb sends three or four rami through the anterior portion of the cribriform plate to the anterior superior portion of the cartilaginous septum. The nerve anastomoses with the nasopalatine and ethmoid nerves

**The Blood Supply of the Nose**—The blood supply of the interior of the nose comes principally from three sources (1) the anterior and (2) posterior ethmoid branches of the ophthalmic artery and (3) from the sphenopalatine artery the terminal branch of the internal maxillary which in turn arises from the external carotid artery. The anterior superior portion of the septum and lateral walls of the nose receive their blood supply from the anterior ethmoid artery the small posterior ethmoid artery supplies only a small area in the posterior superior



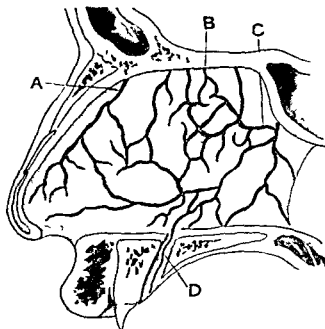
FIG 1



### The Arterial Supply of the Lateral Wall of the Nose

*A* anterior meningeal artery *B* anterior ethmoid artery *C* posterior ethmoid artery  
*D* posterior lateral nasal arteries *E* sphenopalatine artery *F* major and minor palatine arteries

FIG 2



### The Arterial Supply of the Septum Nasi (After Spalteholz)

*A* anterior ethmoid artery, *B* posterior ethmoid artery *C* posterior nasal septum arteries  
*D* anastomosis with major palatine artery

region. Both ethmoid arteries after leaving the ophthalmic cross the cribriform plate and enter the nose by way of the anterior and posterior ethmoid foramina accompanied by the corresponding nerves. The anterior ethmoid artery and nerve indicate to the operator the level of the cribriform plate.

The sphenopalatine artery enters the nose through the sphenopalatine foramen and divides into the posterior lateral nasal arteries which go to the lateral nasal wall and into the posterior septal artery which spreads over the septum (Plate II Figs 1 and 2).

As the posterior lateral nasal arteries are of considerable size it is to be expected that the removal of either the middle or inferior turbinates may be attended by considerable hemorrhage. As a matter of fact the removal of the middle turbinate is usually followed by more or less bleeding for twenty-four hours. There is a free anastomosis between the lateral nasal arteries and the anterior ethmoid artery, hence after the removal of the turbinate bleeding may come from both sources though but one artery is injured.

The posterior septal artery has three main branches: one supplies the posterior, another the inferior, and the other the middle and posterior portions of the septum. These branches which reach the anterior inferior portion of the septum anastomose freely with the superior labial branches to the septum and with the major palatine arteries.

Other branches of the sphenopalatine artery descend in the greater palatine canal to enter the mouth cavity by way of the greater palatine foramen and then spread over the under surface of the palate.

The veins of the nose have a similar nomenclature and follow the course of the arteries. The veins of the vestibule and external structures of the nose communicate with the cavernous sinus by way of the superior ophthalmic vein.

## THE PHYSIOLOGY OF THE NOSE

The functions of the nose are olfactory, phonatory, and respiratory. In man the respiratory function is probably of greatest importance.

**The Sense of Smell**—The olfactory nerve or organ of smell is located in the upper portion of the nasal chambers. The olfactory nerve (Fig. 11) is distributed over the attic of the nose as far downward as the lower margin of the superior turbinate and on the septum over a corresponding area. A knowledge of the area of distribution of this nerve is of practical importance in the diagnosis, prognosis, and treatment of certain diseases of the nose. If there is anosmia or loss of the sense of smell the question arises as to whether the impairment is due to a degenerative change in the nerve itself or to an obstruction to the entrance of the odoriferous particles or emanations to the terminal cells of the olfactory nerve.

The lesions may, however, be intracranial in which case there may be no evidence of either an obstructive lesion or of degenerative changes in the attic of the nose. C. G. Smith<sup>1</sup> in a histopathologic study of 163

<sup>1</sup> Arch. Otolaryngol. 34:533 (September) 1941.

olfactory bulbs in adults found 50 per cent had lost three fifths of their complement of olfactory nerves and 13 per cent had lost all their nerves. This may or may not mean a corresponding degree of impairment of the sense of smell.

**Phonation**—The function of the nose in speaking and singing is important. Many popular public speakers have well-developed nasal resonance while speakers lacking resonance may have difficulty in holding the attention of their audiences. While the initial tone is produced by the vibrations of the vocal cords, the voice is decidedly unpleasant and is not rich in overtones from the resonance chambers of the nose, throat and chest. The nasal chambers and accessory cavities are of prime importance in voice production and any obstruction from swelling of the mucous membrane, deflection, or other lesions of the septum so materially alters the quality of the voice as to make it disagreeable and unartistic.

**Respiration**—The respiratory function of the nose involves two principal activities: conduction of air to the pharynx and the proper preparation of the air for its reception by the lungs.

The generally accepted conception of the inspiratory pathway is a wide curve beginning at the nostril and extending through the olfactory fissure to the posterior choana, thus avoiding to a large extent the inferior meatus. The direction of the air current is determined by the anterior nares, the vault of the nose and the opening of the choana which is larger than the nostril.

There is some disagreement as to the expiratory pathway but in general it is the reverse of the inspiratory route with one probable exception. Because of the relative obstruction at the nares which are the smallest points in the respiratory airway, an eddy is produced which causes some of the air current to flow backward through principally the inferior meatus where it again meets the current rising from the pharynx.

**Cilia**—The nose prepares the air for the lungs by (a) cleansing, (b) tempering and (c) humidifying it. To understand these activities a description of the cilia and the moving blanket of mucus is necessary. The cilia, which are whip-like projections on the free surface of certain epithelial cells, are found throughout the human nose and sinuses except for the anterior one-third (preturbinal area) and the olfactory area. They are also absent in the pharynx. The effective stroke of these cilia is always toward the pharynx. Within the sinuses the cilia carry the secretions to their various ostia.

**The Moving Blanket of Mucus**—Lining the nasal mucous membrane is the thin adhesive slippery and tenacious blanket of mucus. This blanket is moved by the beating of the cilia from the sinuses and nose to the pharynx where it is swallowed or expectorated. It is secreted by the mucous and serous glands (including goblet cells) to the surface of the mucosa. The pH is neutral or slightly alkaline. In the anterior one-third of the nose the mucous blanket is renewed each hour and in the posterior two-thirds and in the sinuses each ten to twenty minutes. By virtue of the tenacity of the mucous coat it is pulled over the non

ciliated preturbinal area by the posterior ciliated portion. Particulate matter which easily passes the barrier of the vibrissæ is almost always caught and firmly held by the mucous coat.

The drainage currents of nasal mucus was first investigated by Yates in 1924 and has since been confirmed by many others. From the non-ciliated anterior one-third of the nose practically all the mucus streaming (i. e., drainage) on the lateral wall is through the middle and inferior meatuses (Hilding).

The streaming of mucus from the posterior two-thirds of the lateral wall of the nose is directed toward the choanæ, with the streams dividing above and below the eustachian orifices. In the olfactory areas of the monkey, which are devoid of cilia as in man, the ciliary flow is perpendicular away from the border (Lucas).

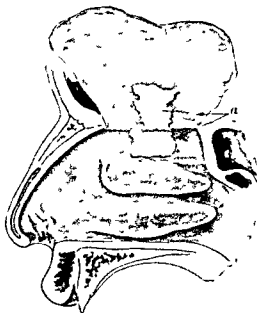


FIG. 11.—Showing the area of distribution of the olfactory terminal nerve cells in the human nose. The triangular flap is the septum turned upward; the area of distribution is limited to the region of the superior turbinate and a corresponding area of the septum, the middle turbinate receiving few or no olfactory cells.

The effect of various drugs topically applied to the cilia has been studied by Proetz, and Lachry and Moore. The normal ciliary activity is of utmost importance to health and the use in the nose of any drug or substance which interferes with the ciliary activity is contraindicated.

As the air passes over and around the turbinates it is warmed or cooled as the case may be to near body temperature. The blood lakes, located primarily in the inferior turbinates, are important in this function, becoming full of blood when the inspired air needs warming and *vice versa*.

It has also been shown that the air in passing through the nasal chambers receives moisture from the nasal mucous membrane. The mucosa

of the lower respiratory tract and the epithelial walls of the air vesicles of the lungs are thus protected from the varying humidity of the atmosphere. In passing through the nose the air is raised (usually) in temperature, thus expanding it and increasing its capacity to absorb moisture. The erectile tissue of the nose, and the glands of the nasal mucosa give off moisture, which is rapidly taken up by the expanded air and carried to the lower respiratory tract, where the glands are much less developed. It has been estimated that approximately one pint of water is thus transferred from the nasal cavities to the lower respiratory tract in twenty-four hours.

The part of the nasal structures which give off most of the water is generally supposed to be the erectile tissue, located chiefly along the free border of the inferior turbinates, and on the posterior ends of the middle and inferior turbinates. It is probable that the mucous glands also give off some of the water. The erectile tissue is under the control of the vasomotor nervous system, which, under normal conditions, regulates the supply of moisture to meet the demands. If the air is dry the cavernous tissue dilates and becomes just active enough to fully saturate the expanded air in the nose; whereas if the atmosphere is humid it is less active. When an obstructive lesion or inflammation is present the cavernous tissue and glands do not respond normally to the atmospheric conditions, hence the air is not properly humidified in its passage through the nose.

## CHAPTER II

### THE SURGICAL CORRECTION OF FACIAL DEFORMITIES

#### RECENT FRACTURES OF THE FACIAL BONES

Due to the automobile and other agencies fractures of the facial bones with involvement of the sinuses and orbits have shown a great increase in recent years. Most fractures involving the facial bones with the exception of the nasal bones extend into one or more of the sinuses or the orbit.

Trauma and secondary deformities of the nose and bones of the face may involve one or more of the following subdivisions: (1) the nose, (2) the nose and forehead, (3) the malar region and orbit, (4) the upper jaw, and (5) the lower jaw.

**Symptoms**—Lymphedema of the soft tissues may occur in fractures involving the sinuses. Displacement of fragments or hemorrhage into the maxillary sinuses are quite frequent. Fractures of the frontal sinus may produce injury to the brain or meninges. Fractures involving the ethmoid sinuses may result in epistaxis and emphysema of the orbital tissues. Cerebral rhinorrhea may be a sequel if the fracture extends to the dura. Fractures involving the sphenoid sinuses may be fatal because of the proximity to the intracranial structures. According to Hagleton fractures through the cribriform plate do not cause meningitis except in cases of a wide defect between the fragments or from the displacement of pieces of bone through the dura.

Surgical shock accompanies the more severe fractures.

**Treatment**—The treatment of compound injuries of the face would depend upon the location, severity and type of injury.

The immediate treatment would demand the maintenance of an adequate airway. This may necessitate pulling the tongue out or holding it out by means of a safety pin and string. A Mosher life saver tube if available may be inserted through the mouth into the larynx.

Hemorrhage should be controlled by pressure or ligation.

The sulfonamides and the antibiotics should be used locally and internally.

Shock if present should be treated by appropriate measures simultaneously with the above.

Debridement or the removal of all devitalized tissue and dirt should be done.

Antitetanus and gas gangrene serum are used in the same manner as for wounds in other portions of the body.

The wounds are sutured so that all anatomic relationships are maintained. Raw areas are eliminated either by suturing or by later skin grafting.

**Fractures of the Nose**—Injuries of the nose may involve one or more of the following structures: (1) nasal bones, (2) frontal process of the superior maxilla, (3) upper lateral cartilages, (4) cartilages of the

ala and (5) septum. The nasal bones may be displaced in an antero-posterior direction, laterally or a combination of directions.

**Treatment.**—With fractures of the nasal bones, care of the lacerations about the nose should receive the first attention. The wound is cleaned and then sutured with small needles and fine silk or subcuticular catgut.

The fractured parts are placed in position by manipulation under gas or local anesthesia.

**Technic.**—If the patient is seen immediately after the accident a local anesthesia consisting of a tampon soaked in a 5 per cent solution of cocaine is very satisfactory. The tampon is placed in the upper region of the nasal fossa (one on each side) and left in place for five or ten minutes. The displaced nasal bones may be lifted into place with the tampon still in the nostril if desired.

After anesthesia a small uterine forceps or a similarly curved, blunt-tipped instrument is inserted into the superior meatus and the fractured bones lifted upward or outward as the case may be. The thumb and forefinger of the left hand are placed over the bridge of the nose to control any over displacement of the bones.

The fractured nasal process of the superior maxilla or fractures of the septum or cartilages (rare) may be placed in their normal position at the time the nasal bones are set.

As a rule intranasal packing is not necessary in recent fractures as the bones seem to stay in place better without than with packing. Intranasal support if used, may be done by means of vaseline gauze packs. Occasionally a suture may be necessary to hold the bridge in position. An aluminum or copper splint, lined with a layer of gauze placed over the bridge of the nose, will give protection to the fractured parts. The splint is held in position by means of adhesive tape. Instead of using the metal splint several layers of adhesive tape placed over the bridge of the nose make an excellent protection.

In some injuries, especially if the patient is unconscious and the bone driven backward, the nose should not be disturbed until a later date.

**Fractures of the Frontal Sinuses.**—The incidence of frontal sinus fractures is about 5 per cent of the facial fractures.

The majority of these cases are without symptoms. A linear fracture through the frontal sinuses may produce bleeding from the nose, and a black eye. If a tear in the dura is present a lumbar puncture may show blood in the spinal fluid. If this occurs from a fracture through the posterior wall of the frontal sinus the mucous membrane may extend into the brain before the fracture unites. As a result bacteria may pass into the arachnoid by direct continuity of tissues.

**Treatment.**—The patients should be confined to bed for a period of from one to two weeks. Blowing the nose and intranasal douches are contraindicated. In fact, no medication or manipulation should be done in the majority of the cases until union has occurred.

With fracture of the inner table of the frontal sinus and a tear in the dura Eagleton advocates placing a layer of skin or fascia lata over the break.

In fractures involving the anterior wall loose fragments of bone may be removed. A crushed frontal sinus wall can be pried out by inserting a chisel into the sinus from just under the orbital border.

**Fractures of the Orbit**—Fractures of the orbit usually involve the malar bone as it constitutes an important portion of the orbital wall. The malar bone may be detached and driven partway into the orbital cavity. Marked edema and contusion of the overlying tissue are invariably present. If the infraorbital nerve is injured anesthesia hyperesthesia or paresthesia of the cheek may occur.

**Fractures of the Malar Bone**—Fractures involving the malar bone with associated injuries of the orbit or nose are frequent. The malar bone is usually depressed producing a change in facial contour.

**Treatment**—Various methods have been used to reduce the displaced fragments such as elevation by means of a silver wire suture passed around the depressed bone, a hook or bullet forceps to pull the depressed fragment into position, a sound introduced through a naso-antral window, the depressed zygomatic arch elevated by inserting a flat periosteal elevator through an incision above the first molar tooth, a screw inserted into the bone through a stab incision. Gill<sup>1</sup> uses a special grasping forceps to seize the bone and move it into its normal position. A heavy towel clip may be used for the same purpose. The hook is passed through a small nick in the skin below the fragment. The fragment is then elevated until the proper approximation is reached. The hook may be used to hold the fragment in elevation until fixation has taken place.

If the lower margin of the orbit is defective a curved piece of rib will replace the lost bone (New).

*Comminuted fractures involving the orbit as well as the malar bone* may require special retaining appliances such as an interdental splint and a plaster head cap or wiring the fractures by means of a heavy silver wire suture which is allowed to remain *in situ*.

**Fractures of the Superior Maxilla**—Fractures of the upper jaw may result in a displacement backward or lateral of the upper jaw and teeth. The maxillary sinus is usually involved filling with blood.

**Treatment**—With a transverse fracture of the superior maxilla with separation from the skull it is necessary for the dental surgeon to make splints and appliances to reduce and hold these fractures in place. If the alveolus only is fractured an interdental wiring of the teeth is sufficient. Rubber bands attached to crowns about the teeth drawing the displaced fragment downward may be used with satisfactory results in many cases.

Loose non vital fragments and loose teeth should be removed. If the fracture is not extensive a four tail bandage may hold the lower jaw against the upper jaw in correct alignment. In edentulous cases a plate can be used attached to a suitable headgear.

The blood or hematoma within the maxillary sinus usually drains or is absorbed without surgical intervention.



**Complications.**—Following this type of fracture numbness of the cheek may persist for months. This may be followed by a neuritis. If improperly set, malocclusion of the teeth may occur. A purulent dacryocystitis sometimes follows. Disturbance of vision may follow extensive fractures. Chronic sinusitis develops at times. Deformities may result in some cases from scar contraction. Sequestra may form and be extruded.

**Fractures of the Mandible.**—In fractures of the lower jaw, wiring the teeth together by means of silver wire may be sufficient to give good results. At times interdental vulcanite splints attached to casts on the head are necessary, especially if the teeth are absent from a posterior fragment. A coat hanger wire suspended from the cast extends to the mastoid region. A silver wire is passed through a hole in the angle of the lower jaw and this wire is then attached to the wire from the cast on the head. The fragment is then placed in proper alignment.

Any teeth that are in the line of fracture should be removed to produce a proper union. "Fractures of the lower jaw, with loss of tissue, if function is good, do not require any treatment. However, if tissue has been lost and displacement of the jaw interferes with mastication, bone grafts should be employed" (New). If teeth are present near the bone graft they should be removed and further work delayed for three months to guard against any infection from the sockets. A sliding graft may be used in which a portion of the jaw is slid across to fill in a narrow space or an osteoperiosteal graft if there is a slight loss of tissue. If more than 2 to 2.5 cm. of tissue is lost, a bone graft from the ilium should be used (New).

## OLD FRACTURES AND DEFORMITIES OF THE FACIAL BONES

In plastic nasal surgery local infiltration anesthesia has superseded general anesthesia to a great extent. Block anesthesia may be used as it is followed by less swelling of the operative fields. If the latter method is chosen both infraorbital nerves are injected with 1 or 2 per cent novocain solution and 20 per cent cocaine is applied to each sphenopalatine ganglion. A 5 per cent cocaine solution is applied to the septal mucosa. The columella is infiltrated, especially the base. A 6-grain ampule of amytal may be given one-half hour before operating.

**The Depressed Nasal Bone.**—The depressed nasal bone should be fractured from its attachment and reset in its normal position. This should be done two or more weeks after the submucous resection if necessary. The technic is as follows:

An intranasal incision is made with a small scalpel through the mucous membrane of the outer and anterior wall of the nose at the inferior border of the nasal bone (Fig 12). A semisharp septum periosteal elevator is then introduced through the incision, and the skin and periosteum over the nasal bone stripped loose.

The ring forceps, Steel, Asch, or other stout septum forceps is introduced into the nostril thus prepared, and one blade insinuated through

the incision and between the skin and nasal bone while the other remains free in the nose (Fig. 13)

The nasal bone is firmly grasped between the blades of the forceps, and rotated upon the axis of the blades and the nasal bone completely fractured from its attachments. Instead of the forceps a chisel may be used for separating the nasal bones. A small chisel is placed a little anterior to the outer extremity of the pyriform aperture and a small bone incision made in a direction parallel with the nasal bridge. The chisel is then placed at about the root of the nose and over the body of the nasal bone the chisel being perpendicular to the long axis of the nose and another bony cut made. The fracture can then

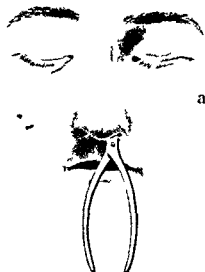


FIG. 1. The intranasal incision at the tip of the left nasal bone. One blade of the steel forceps is inserted through this between the skin and the nasal bone the other grasps the tissue anterior to the middle ethmoidal sinus.

FIG. 13.—The steel forceps for grasping the nasal bone (a) to fracture it. The position of the forceps is shown to be anterior to the middle ethmoidal sinus.

easily be made by a few sharp taps on the side of the nose. The scar on the skin is small.

If the septum is out of the center line and a submucous resection has not been done an attempt may be made to centralize the septum by means of the Asch septum forceps.

The nasal bone should be reset in its normal position and held there by means of a copper splint cut and shaped to fit from sheet copper which is placed on the nose and held in place by adhesive tape.

If there is a marked spreading or broadening of the nasal processes of the superior maxilla this may be helped by sawing or chiseling through their bases and in fracturing them medially after elevating the periosteum.

If necessary a supportive graft of bone or cartilage may be inserted at a later date to correct any remaining defect.

**Saddle-back Nose.**—Saddle nose may be the result of syphilis (congenital or acquired), trauma, septal operations, septal abscess, ozena, nasal lupus, cretinism, heredity, etc.

Various substances have been used for the correction of depressed nasal deformities or saddle-back nose. The ones usually employed are cartilage, bone and ivory.

**Cartilage.**—Cartilage withstands infection and absorption fairly well, is readily accessible and can be moulded and handled with ease. It is usually obtained from the auricle, septum and the lateral or costal cartilages. The septal cartilage tends to absorb in part and when used an overcorrection is indicated. It seems to be established that rib cartilage seldom absorbs but undergoes slight calcification. Cartilage



FIG 14 —Congenital saddle nose due to cretinism



FIG 15 —Traumatic saddle nose

seems to be preferable where the defect is small enough to permit its use. A complete correction of a pronounced nasal defect by rib cartilage *may be impossible on account of its curvature in the costal arch, which prohibits the removal of a long and straight transplant.* In these pronounced nasal deformities an ivory prosthesis offers a good substitute.

When it is necessary to take a graft most authors advise the selection of the seventh, eighth or ninth rib. Roy recommends the use of the cartilage of the first floating rib as being easily resected and with less after discomfort. For reconstruction of a nasal depression involving the bridge, as well as the tip of the nose, an angular graft united at the knee by a strip of perichondrium is used frequently. Maliniak<sup>1</sup> recommends separate supports for the dorsum and columella.

<sup>1</sup> Arch Otol, 17, 649, (May) 1933

**Bone**—Bone grafts may be obtained from the rib crests of the tibia ilium free vertebral border of the scapula or from the turbinates

Carter's radiographic studies indicate that the outer layers of bone transplanted into the nasal tissues remain unchanged whereas the central portions being more remote from the circulation reveal definite signs of absorption which he thinks is probably due to impaired nutrition Ferris Smith Sheehan Davis and Gillies believe that the bone grafts eventually absorb and are replaced by a deposit of fibrous tissue the clinical results depending on the amount of the new formed tissue The presence or absence of the periosteum does not seem to be a factor in determining the resorption Most plastic surgeons remove the perichondrium as its retention causes the cartilage to curl in some cases

In order that the bony graft may succeed it is essential to place it in contact with the nasal bones and to perform the operation in an aseptic manner

**Ivory**—In 1918 Joseph of Berlin advocated ivory for the correction of saddle nose This organic material very much resembles human bone and is generally well borne after having been encapsuled by fibrous tissue In rare circumstances it produces a mechanical irritation and is not tolerated It is being used less and less

The pseudo ivories being entirely foreign to human bone are ejected sooner or later Ivory is best sterilized by boiling from twelve to fifteen minutes

**Celluloid**—The present opinion seems to be that celluloid is irritating and is sooner or later eliminated

**Paraffin Injection**—The use of paraffin is obsolete at the present time due to the various complications attending its use such as paraffinoma embolism etc

## RHINOPLASTIC RECONSTRUCTION

Rhinoplastic surgery or the correction of nasal defects either esthetic or functional has received much impetus in recent years by the work of Metzenbaum<sup>1</sup> White<sup>2</sup> Peer<sup>3</sup> Lomon<sup>4</sup> Salinger<sup>5</sup> Scher<sup>6</sup> Ersner<sup>7</sup> Seltzer<sup>8</sup> and others Many defects or abnormalities congenital or acquired singly or collectively are amenable to correction by portions of or modifications of this rhinoplastic procedure This operation is indicated in many cases of septal deviations associated with external deformities in which the nasal function may not be restored completely except by some rhinoplastic reconstruction in addition to the classical submucous resection of the septum

<sup>1</sup> Arch Otolaryngol 9 252 (March) 1909

<sup>2</sup> Arch Otolaryngol 11 415 (April) 1930

<sup>3</sup> Arch Otolaryngol 25 475 (April) 1933

<sup>4</sup> Surgery of Injury and Plastic Repair Baltimore Williams & Wilkins Company 1939

<sup>5</sup> Arch Otolaryngol 29 50 (March) 1939

<sup>6</sup> Arch Otolaryngol 34 30 (August) 1941

<sup>7</sup> Arch Otolaryngol 39 46 (June) 1944

<sup>8</sup> Arch Otolaryngol 40 433 (December) 1944

**Technic-Anesthesia**—Anesthesia of the septum is obtained as described for the submucous resection of the septum. Anesthesia of the external portion of the nose results from blocking the infratrochlear, infraorbital, external nasal and interior palatine nerves. A 1 or 2 per cent solution of procaine hydrochloride with 5 to 10 drops of a 1 to 1000 solution of epinephrine hydrochloride to the ounce is injected intranasally.

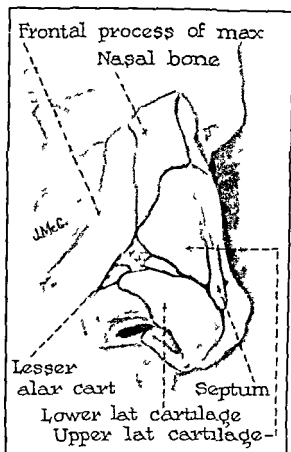


FIG. 16—The external framework of the nose

The infratrochlear nerve is blocked by injecting about 0.5 cc. of the solution into each side of the nose by means of a 2 inch, 24 gauge needle through the nasal fold (limen nasi) to the nasal root. The needle is inserted above the perichondrium and the periosteum.

The infraorbital nerve is blocked by injecting bilaterally 0.5 cc. of the solution through the extreme outer margin of the piriform opening to a point intersected by a vertical line drawn 1 cm. lateral to the inner canthus of the eye and an oblique line extended from the columella to the outer canthus of the eye.

The nasopalatine and anterior palatine nerves are blocked by bilaterally injecting about 0.5 cc. of the solution at the anterior nasal spine along the floor of the nose to the incisive foramen. The fluid is injected in all these positions during withdrawal.

**Uncovering the Nasal Framework.**—The nasal framework is uncovered by first making bilateral incisions through the mucosa and tissue between the upper and lower lateral cartilages. A straight pointed

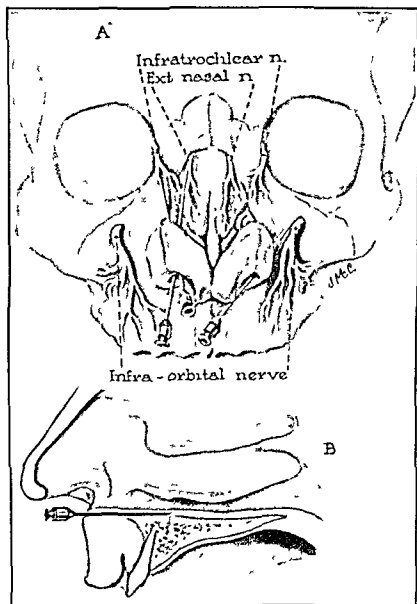


FIG 17—Nerve blocking for rhinoplastic reconstruction. A, Blocking the nerves supplying the external surface of the nose. B, Position of the needle for blocking the anterior palatine nerve.

double edge knife or small periosteal elevator is inserted through the incisions between the skin and perichondrium of the upper lateral cartilages to the root of the nose. The knife or elevator loosens the periosteum and skin from the dorsum of the nose. Injury to the lacrimal sac should be avoided. The perichondrium covering the upper lateral nasal cartilages is not elevated.

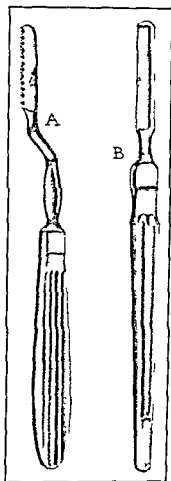


FIG. 18—*A* Joseph bayonet saw for removing the bony nasal hump. *B* Straight blunt pointed knife for separating the columella from the cartilaginous septum.

**Exposure of the Anterior Edge of the Septum**—The caudal or anterior exposure of the cartilaginous septum is made by inserting a button end knife through the left intercartilaginous incision above the upper lateral cartilage perichondrium but below the nasal bone periosteum to the nasofrontal suture. The knife is swept obliquely downward across the dorsum of the nose until the right intercartilaginous incision is reached. The knife, held at a right angle, then divides the superior two-thirds

of the membranous septum close to the anterior margin of the cartilaginous septum. The separation of the inferior one-third of the membranous septum is made by means of straight Mayo scissors.

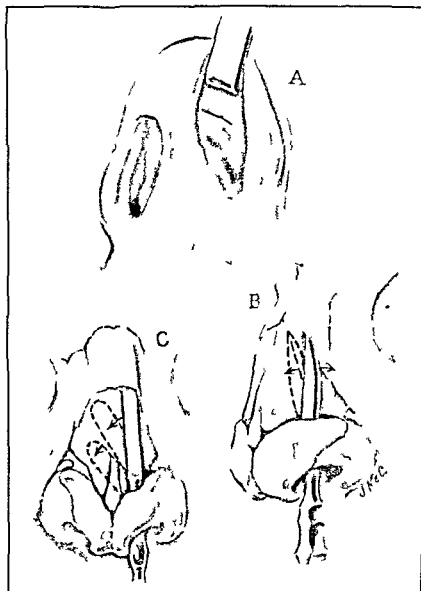


Fig. 10. A. An incision through the membranous tissue between the upper and lower cartilages. B. Lie at the base of the pericardium on the upper lateral cartilage. C. Lifting the pericardial space on the lower part of the nose.

**Removal of the Excessive or Deflected Portion of the Nasal Septum —**  
After the caudal portion of the cartilaginous septum has been freed the septum is freely mobilized and can be pushed to one side in full



view. Any excessive portion for shortening of the nose can be trimmed away or any deflected portion removed as in the conventional submucous resection. Seltzer<sup>1</sup> repositions the deflected portion of the septal cartilage by incising a narrow, vertical strip from the dorsum to the base through the buckled portion, then separates the anterior portion from the base by an anteroposterior incision along the nasal floor. The anterior, free

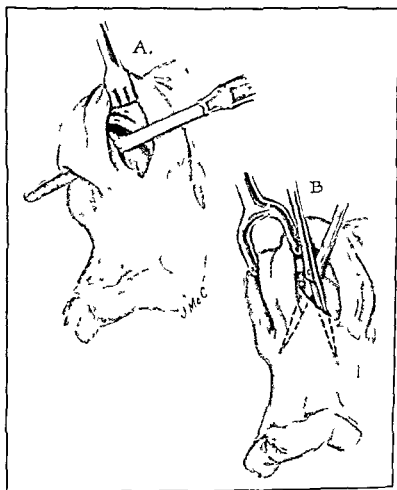


FIG. 20.—The exposure of the anterior edge of the septum. *A* A button end knife divides the superior two-thirds of the membranous septum. *B* Straight Mayo scissors separate the inferior one third.

segment of cartilage is then placed in a central position and sutured in place. Two sutures are placed in the posterior incision and the anterior edge is sutured to the columella. The septum is held in position by carbowax gauze.

<sup>1</sup> Arch. Otolaryngol., 40: 433 (December) 1944.

**Shortening of the Upper Lateral Cartilages** — If the septum has been shortened very much it may be necessary to remove the lower portion of the upper lateral cartilages. The shortened septum and columella are re-approximated and the projecting portions of the cartilages are severed by means of angulated scissors.

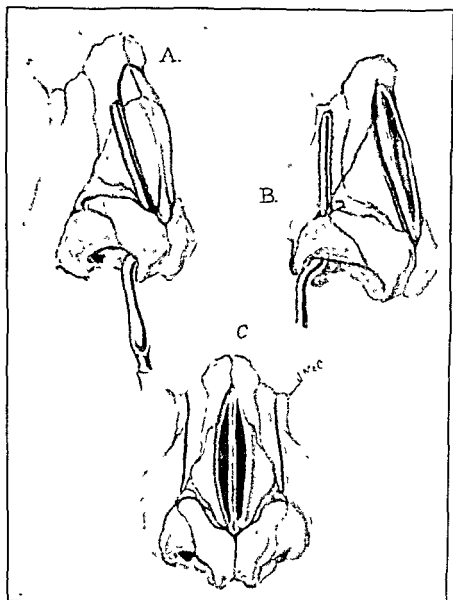


FIG. 21 — Removal of the hump and narrowing of the nose. A, A bayonet saw separates the excessive portion of the cartilaginous and bony hump. B, The bayonet saw separates the stumps of the nasal bones. C, The bilateral cuts completed, preparatory to infracturing the severed portions.

**Removal of the Hump** — After the nasal framework has been uncovered a bayonet saw is introduced through the incision in the nasal plica up to the nasofrontal suture but beneath the periosteum of the nasal bone. The desired level of the nasal bones and cartilages are severed at exactly the same level on each side. If the inferior anterior portion of the hump is still attached it is separated by means of a short button knife. The severed portion is removed through the vestibular incision by means of a nasal forceps. If any irregularity remains in the two sides they are reduced by filing with a rasp.

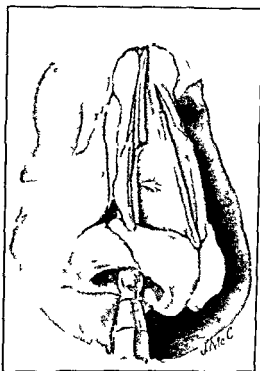


FIG. 2. Removing a segment of bone from the nasal bridge on the broad side preparatory to outfracturing with the Roman guarded chisel.

**Narrowing of the Nose** — The flat dorsum of the nose left after severing the hump is corrected by sawing the stumps of the nasal bones at or near their maxillary sutures and removing the solid wedges of bone at their roots. The nasal bones are fractured outwardly at their nasofrontal articulation by means of the Roman guarded chisel. The fractured nasal bones are then pushed together and held by a stabilizing splint. If one side of the nose is flatter than the other it may be necessary to remove an extra segment of bone from the broad side.

The upper lateral cartilages are approximated with one or two very fine (6-0) chromic catgut sutures through the upper or ventral margins.

**Modeling of the Lower Lateral Cartilages** — If the lobules of the nose formed by the lower lateral cartilages are relatively too wide or if the

tip of the nose has a projecting, bulbous appearance it may be advisable to excise a portion of these cartilages.

The lower lateral cartilages are exposed by incisions along their lower margins. The angle of each cartilage is freed from the overlying struc-

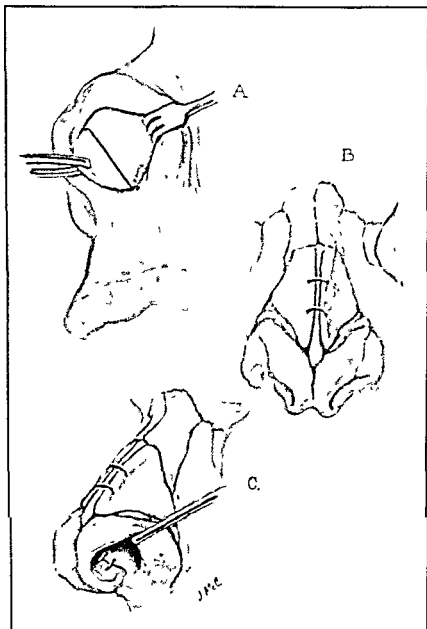


FIG. 23—Shortening the nose. *A*, Excising the excess portion of the caudal edge of the cartilaginous septum. *B*, If the nose has been shortened very much it may be necessary to trim the lower edges of the upper lateral cartilages. The cartilages are held in place by one or two very fine sutures. *C*, The shortened septum and columella are re-approximated.

tures (the perichondrium is not elevated) so that the lobule is free. The angle of the cartilage is partially drawn out and trimmed to suit each individual case.

**Dressing and After-care** — The nose is held in place by the external application of Stent's composition moulded over soft flannel. The splint is retained by adhesive strips. The nasal cast is removed in from two to five days.

**The Correction of Saddle back Nose** — **Preparation** — A plaster of Paris cast is taken of the face and a positive prepared. The defect in the cast is filled with wax in order to obtain the exact size and shape of the implant. The thickness of the skin is deducted from the pattern in order to obtain an implant accurately fitted into the defect.

Local anesthesia preceded by the administration of from 3 to 6 grains of sodium amylal and the immediate injection of morphine and hyoscine before the operation is satisfactory as a rule. Anesthesia of the external portion of the nose is obtained by blocking as described for Rhinoplastic Reconstruction.

**Incision** — A vertical incision is made in the under surface of the nasal tip. A subcutaneous tunnel is undermined by means of long scissors extending as far as necessary toward the infraglabellar notch. The dissection should not extend beyond the limits in which the implant will lie otherwise a displacement will occur. Undue pressure of the implant upon the skin or mucous membrane should be avoided.

Gillies makes vertical incisions in the vestibule of each nostril near the junction of the skin and mucous membrane. These incisions are united by dividing the columella at its attachment to the upper lip. The columella is then dissected free, seized with a suitable instrument and drawn upward to expose the free border of the columella above which a tunnel is effected toward the nasal arch. The incisions may be made also as described for Rhinoplastic Reconstruction.

If a slight depression of the bridge is associated with a hump the excised cartilage or bone from the hump may be used for filling in the depression. The saddle nose without a hump can be corrected by the use of either cartilage or bone. When a large graft is required bone seems to offer the best material.

If the saddle nose is abnormally wide, with broad nares it is necessary to make the tip and bridge narrower and to correct the nares as described in Rhinoplastic Reconstruction. If the lower part of the nose is flattened or the lower cartilage of the nose collapsed two interlocking pieces of ivory as advocated by Salinger may be used.

The columella incision is closed with fine silk or horsehair sutures which are removed after two or three days. To assure good immobilization of the implant a thin lead or copper splint may be applied externally.

**The Long or Drooping Nose** — This type of nose is occasionally seen. The resection of a wedge-shaped piece of the nasal septum through the nasal orifice is satisfactory in most cases. The technic is described in Rhinoplastic Reconstruction.

**The Twisted or Crooked Nose**—This type of deformity may be due to the congenital maldevelopment of the structures of the nose and face, but it is generally caused by external violence to one side of the



FIG. 24—Traumatic lateral displacement of the nose to the right a depressed left nasal bone

nose which results in an irregular lateral displacement of the septum and tip. The nasal bone upon the side receiving the blow may be displaced laterally or depressed.

The technic for straightening the twisted nose and associated irregularities is described in Rhinoplastic Reconstruction.

**The Hump Nose**—The hump nose, also known as the aquiline or hook nose, may be congenital or traumatic. It is one of the most common nasal abnormalities.

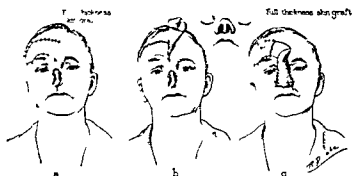


FIG. 25—*a* Flap on the forehead with the full thickness skin graft used as a lining for the skin that will form the body of the nose. *b* flap elevated about to be brought down in position to reconstruct the nose. *c* full thickness skin graft in the wound in the right side of the forehead and the flap in position on the nose (Gordon B. New).

The congenital type of nasal hump is due to an overdevelopment of the bony bridge and the quadrilateral septal cartilage and at times of the external cartilages of the nose.

The traumatic type of deformity is usually confined to the upper two-fifths of the nasal base. Both types may be associated with other abnormalities such as a deviated septum or columella, twisted nasal tip or laterally placed nasal bones.

Treatment is by the surgical correction of the abnormality as described for Rhinoplastic Reconstruction.

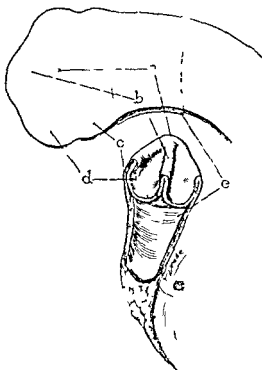


FIG 2 —Detail of forehead flap. *a* point that forms the tip of the nose. *b* columella. *c* covering of the ala. *d* lining of the ala. *e* full thickness skin graft which lines the upper portion of the nose (Gordon B. New).

**The Broad Nose** —The usual procedure in narrowing the nose is to fracture or separate the nasal bones and the frontal processes of the superior maxilla as described in Rhinoplastic Reconstruction. They are then moved towards each other and away from the frontal bones. This will narrow and lift up the nose.

**Deficiency Defects of the Nose** —With a loss of a portion of the nose it is first necessary to determine whether there is a deficiency of covering skin, lining mucous membrane, and supporting bone or cartilage or a combination of two or more of these. If two or more of these tissues are wanting and only one is replaced at operation, the result is not likely to be very successful.

For deficiencies of skin covering, pedicled flaps may be obtained from adjacent parts.

Lining of the nose may be accomplished by folding in the distal end of the forehead flap or the skin from the margins of the defect may be used by inverting the pedicled skin flap (Figs 25 and 26). Thiersch (I-sser's epithelial inlay) or full-thickness skin grafts may be used.

Supporting substances such as ivory, bone and cartilage are used. In this country costal cartilage seems to have the most advocates. In Germany the osteoperiosteal fragments taken from the leg are commonly employed. Costal cartilage is not difficult to obtain and is easily trimmed to suitable shape and rarely undergoes absorption when embedded in soft tissues.

**French Method** — In the procedure known as the French method the flaps are taken from nearby portions of the cheek and transferred to the nasal defect. It has the disadvantage of producing an additional scar.

**Italian Method** — In the Italian method a flap of skin is taken from the arm. Its free end is sutured into the nasal defect, the forearm being fixed to the head by means of a plaster cast until union occurs. At a second operation the arm pedicle is severed and the flap fashioned to form the nose.

R. H. Ivy reports a modification of the Italian method used by Ferris Smith which presents many advantages. "A tube pedicle is first prepared on the arm, its distal end being just above the elbow. At a second operation the lower end of the pedicle is severed and brought up to the facial defect. It is maintained by fixing the arm in the Velpeau position with plaster of Paris, the head being slightly inclined toward the shoulder."

Ivy advocates the forehead as the best source of a pedicled flap to supply skin for the nose. "A flap with a pedicle either at the inner third of the eyebrow, nourished by the angular and supra-orbital arteries, or at the temporal region, nourished by the superficial temporal artery, is well supplied with blood, is easily maintained in position and the skin has a texture very similar to the normal skin of the nose. Flaps of sufficient size can be obtained from the forehead to supply the entire covering of the nose as well as lining the alæ and forming the columella."

**Esser's Epithelial Inlay** — Esser's method of applying Thiersch grafts to denuded surfaces of the nose has given great impetus to plastic surgery. A Thiersch graft is introduced by means of a moulded dental compound shaped in the form of the cavity to be lined. The Thiersch graft is wrapped around the mould and left in place for about ten days. When removed, and if successful, it leaves the cavity epidermized. It is of great value in replacing the missing nasal mucous membrane in the correction of the syphilitic nose. In the repair of the syphilitic nose there is undoubtedly a greater risk of gangrene occurring in a flap because the presence of a syphilitic endarteritis interferes with the proper nourishment of the flap.



**The Short or Absent Columella** — Dieffenbach's method consists in making two vertical parallel incisions through the entire thickness of the middle of the upper lip including the mucous membrane thus forming a strip of tissue about 1 cm wide. The mucous membrane representing the vermilion border of the lip is removed, and this freshened end is then sutured to a raw surface made just beneath the tip of the nose. The raw sides of the strip are partially obliterated by suturing the skin and mucosa together.

The edges of the defect in the lip are brought together with sutures. A short columella may be lengthened by detaching the septum from

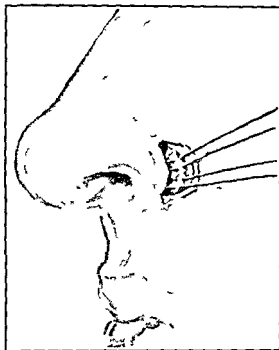


Fig. 27. Lateral transfixation of the alæ for atresia or collapse of the alæ nasæ.

the columella then extending the two incisions into a portion of the upper lip. The columella and the cut portion of the lip is then sutured to the septum in a higher position than the columella formerly occupied.

**Atresia or Collapse of the Alæ Nasi** — Atresia or collapse of the wings of the nose is sometimes associated with prolonged nasal obstruction and mouth breathing. The condition may be hereditary or due to senile changes. Cinelli<sup>1</sup> attributes the narrowing to a muscular imbalance or atrophy of the dilator and constrictor muscles of the nose and to atrophy of the upper lateral and alar cartilages.

The nasal orifices are greatly narrowed, often mere slits, and the alæ are flaccid and collapse under inspiration. Under normal conditions the alæ dilate and are firm and resilient.

<sup>1</sup> Arch. Otolaryngol. 33: 683 (May) 1941.

**Treatment**—If the collapse is due to unilateral nasal obstruction the cause of this obstruction should be removed. In some instances this is followed by a cessation of the collapse especially if the condition is of comparatively recent occurrence. In older cases the collapse of the alæ persists.

For the surgical correction of this condition Cinelli<sup>1</sup> advocates a lateral transfixation of the nares. This is accomplished by making a semilunar incision in the nasolabial fold with resection of a segment of the cutaneous, subcutaneous and muscular layers down to but not including the nasal mucosa. A long straight needle with 00 chromic catgut is passed outwardly through the nasal mucosa at the highest point of the incision. The same suture is re-inserted just below the first. The ends of the suture are then fixed laterally near the posterior border of the excised area. A suture is placed in the lower portion of the incision similar to the first. The sutures are then tied resulting in an enlargement of the collapsed nares. The skin is closed with dermal sutures. Packing or external dressing is not necessary.

**Bi-fid Nose**—A bi-fid or cleft nose is a congenital deformity characterized by a median furrow or cleft in the dorsum. It may occur alone or with other facial deformities.

The abnormality is due to improper fusion of the pre-scutous and pre-cartilaginous tissues of the embryo.

The treatment is by some form of rhinoplastic surgery in which the skin of the dorsum of the nose is undermined and cartilage or bone inserted. Associated nasal deformities may be corrected by methods similar to those described under Rhinoplastic Surgery.

**Old Depressed Fractures of the Frontal Bone**—Old symptomless depressed fractures of the frontal bone should be let alone unless the depression is limited to the outer wall. In this event the outer wall may be forced outward into as nearly correct alignment as possible. Any scars that may be present should be removed.

Instead of entering the frontal sinus the depressions may be filled subcutaneously with a muscle, fat or a flat cartilage graft. The cartilage graft is inserted through a small incision.

If a portion of the supra-orbital ridge is missing a bone graft from the crest of the ilium is satisfactory. The graft is shaped to fit the bony loss and held in place by catgut or wire.

<sup>1</sup> Arch. Otolaryngol. 31: 53 (January) 1910.

## CHAPTER III

### DEFORMITIES AND DEVIATIONS OF THE NASAL SEPTUM

**Etiology.**—Irregularities of the nasal septum may be due to trauma in a few instances, especially the cartilaginous portion. The vast majority of both cartilaginous and bony deviations and deformities, however, seem to be developmental in origin. The cause is unknown. Many theories have been expressed, but none of them proven. They are common in the Caucasian but uncommon in the more primitive races. Men are affected more frequently than women. They become clinically manifest during adult life rather than in childhood. They are frequently associated with the high or "Gothic" arch of the bony palate, although the reverse may be true. The high "Gothic" arch is normally present in infants. It is possible the lack of descent or the lack of broadening of the hard palate would crowd the normally developing septum, causing it to extend out of the mid-line.

It would seem these developmental irregularities of the septum would have some relationship to evolutionary changes involved in the expanding brain cavity and the diminishing facial structures. At least there is a disproportionate relationship between the growing septum and its bony framework.

**Types.**—The types and locations of the various deviations, spurs, ridges, etc., of the nasal septum have to a considerable degree lost their clinical significance insofar as treatment is concerned, since the perfection of the submucous resection of the septum has been accomplished, and so many types of septal malformations are found to be amenable to it.

**Cartilaginous Deviations.**—When the deformity is limited to the cartilaginous portion of the septum it is one of three types, viz :

(a) A deflection of the anterior portion generally known as the columnar cartilage (Fig 28). The antero-inferior border of this cartilage is turned outward into the vestibule of the nose and obstructs the respiratory passage. This type of deviation is not as serious in its consequences as those that obstruct the nasal chamber in the region of the middle turbinate, as it only interferes with the ventilation of the nasal chamber and accessory sinuses, the drainage being unimpaired, except insofar as it depends upon the mechanical aid of the air current in propelling the secretions to the nasopharynx.

(b) An angular deviation in an anteroposterior direction is serious in proportion to its proximity to the middle turbinate. If it is limited to the region of the vestibule or the inferior turbinate it is of less clinical importance, though its removal is still indicated. If it obstructs both the middle and the inferior meatuses its removal is of greatest importance, as it interferes with both the drainage and ventilation of the nasal chamber and the accessory sinuses of the nose.

(c) A perpendicular deviation of the cartilage only interferes with the ventilation without blocking the drainage of the secretions except anteriorly, which is inconsiderable.

**Osseous Deviations** — For clinical purposes osseous deviations of the septum may be divided into three types.

(a) A bony ridge along the upper border of the maxillary and palatine crests is frequently present. The direction of this deformity is backward and upward usually beginning anteriorly about  $\frac{1}{2}$  inch from the border of the inferior portion of the nasal opening near the floor of the nose. A ridge in this location does not necessarily obstruct the normal



11-25 — Deviation of the anterior portion of the septal or columnar cartilage which may be removed through Hays' incision by sharp dissection.

inspiratory tract (middle and superior meatuses) nor does it greatly interfere with the drainage of the secretions. It does, however, encroach upon the inferior turbinate and this causes irritation of this important physiologic organ and produces a sense of stuffiness of the nose. It interferes also to some extent with the posterior drainage of the secretions. It also projects to some extent into the respiratory pathway and forms a favorable place for the desiccation of the secretions. Crusts are, therefore, generally found upon the anterior extremity of the ridge and in blowing the nose become detached tear the epithelium and give rise to epistaxis.

(b) The perpendicular plate of the ethmoid bone is often convex or cup-shaped and impinges upon the middle turbinate upon the side of convexity. This is perhaps, one of the most serious obstructive lesions of the septum, as it obstructs both the drainage and the ventilation of the superior meatus and of the frontal, ethmoid and sphenoid cells.

(c) The combined deviation including the ridge along the crest of the vomer and the convexity of the perpendicular plate of the ethmoid (Fig. 29) is a very common type of septal deformity and often calls for correction at the hands of a surgeon.

(d) There are still other deformities of the osseous septum, as the so-called spurs on the anterior portion, which in reality are composed of the nasal crest and cartilage in combination though they may be true osteomata.

**Indications for Operation** —The indications for the correction, or the removal, of obstructive deviations of the septum are based upon the following considerations

If the deviation of the septum does not interfere with (a) the functional activity of the inferior turbinates, (b) the ventilation of the middle and superior meatuses and the accessory sinuses, (c) the drainage of the same areas or (d) with nasal respiration it should not be subjected to surgical treatment. In other words, deviations of the septum should never be corrected simply because they are departures from the median line of the nose, but only when they obstruct ventilation and drainage or interfere with the function of the turbinates or nasal mucosa.



FIG. 23 — A compound deviation of the septum. The upper deviation is of greater clinical importance as it blocks the ventilation and drainage of the sinuses.

If a ridge along the crest of the vomer is so prominent as to crowd the inferior turbinate, or if it extends forward into the vestibule far enough to partially obstruct the inspiratory current of air, it should be removed. The same is true in reference to anterior angular deflections of the cartilaginous septum.

If the deviation is higher up, in the region of the middle turbinate, and interferes with the ventilation of the superior meatus and the accessory sinuses draining into it, it should be corrected.

**Contraindications to the Septal Operation** —Contraindications are untreated syphilis, diabetes, advanced tuberculosis, acute rhinitis, acute sinus disease, acute middle-ear disease, atrophic rhinitis, marked hyperplastic rhinitis, and children under sixteen years of age whose facial development is not complete.

### THE SYMPTOMS OF DEVIATIONS OF THE SEPTUM

A common symptom of nasal obstructions is a sense of fulness, either in the lower or upper portion of the nasal chambers according to the location of the deviation. If, for instance, the deviation impinges upon the inferior turbinate there is a sense of stuffiness or fulness in the lower

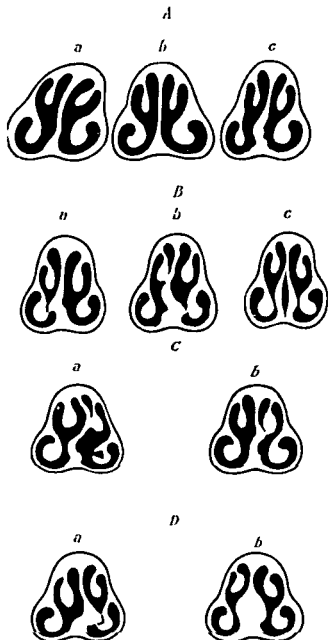


FIG. 30.—A Types of non-obstructive septa. *a* deviated from the median line. *b* normal straight septum in the median line. *c* deviation of the lower portion of the septum with a concavity in the left nasal chamber but with compensatory hypertrophy of the left inferior turbinate.

B Types of obstructive septa. *a* ridge pressing against the inferior turbinate. *b* ridge pressing against the left inferior turbinate and a convexity higher up on the right side obstructing the olfactory fissure on that side. *c* a split septum causing double obstructive convexity of the septum.

C *a* An E-shaped septum causing obstruction in the inferior portion of the nasal chamber on the right side and the superior portion of the chamber on the left side. *b* a high angular deviation of the septum causing obstruction of the olfactory fissure of the left side.

D *a* Marked deviation of the septum along the crest, the vomer wedged firmly against the left inferior turbinate. *b* abscess or hematoma of the septum obstructing both nasal chambers.

portion of the nose whereas if it is in the region of the middle turbinate there is a sense of stuffiness or pressure through the bridge of the nose between the eyes

If the obstruction in the region of the middle turbinate is great enough or has given rise to a congestive inflammation in the anterior ethmoid cells there may be pain upon pressure at the inner angle of the orbit under the floor of the frontal sinuses

Frontal headache is frequently present in high deviations and is most severe in the morning upon awakening. If of ocular origin it subsides at night and recurs during the day while using the eyes.

The nasal secretions may be changed in character and quantity. If a chronic congestive inflammation of the lower portion of the nasal mucous

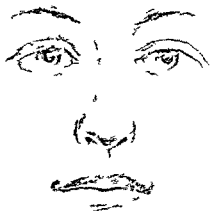


FIG. 31 — A traumatic deformity of the external nose and of the septum. The straight dotted line indicates the median line of the nose while the curved one indicates the deviation of the septum.

membrane is present the secretions are heavier than normal and expulsion is only accomplished by blowing the nose. If the obstruction is in the middle turbinate and ethmoid regions and a simple inflammation is present in the ethmoid cells the secretion is sometimes watery in consistency though it may be mucoid and quite acrid in character. Associated signs of this type of secretion are the reddened and irritated appearance of the mucosa and a fissure or eczematous eruption of the margins of the nostrils and the upper lip. A postnasal or epipharyngeal dropping is usually present.

Intermittent stenosis is usually present in those cases in which there is an anterior deviation which does not completely block the nasal passage.

A permanent partial or complete stenosis of the nasal respiration on the affected side is present if the deviation from normal is marked.

External deformity of the nose is often indicative of a corresponding deviation of the septum (Fig. 31).

## CHAPTER IV

### THE SURGICAL CORRECTION OF OBSTRUCTIVE LESIONS OF THE SEPTUM

THE submucous resection of the septum has largely replaced all former methods of straightening the septum hence the descriptions and illustrations of the older and more or less obsolete methods which appeared in former editions of this work have been omitted from recent editions.

#### THE SUBMUCOUS RESECTION OF THE SEPTUM

**Position of the Patient** — The patient may be placed in either the sitting or the reclining posture. Most operators will probably prefer the sitting posture in an ordinary office chair though the reclining posture may become necessary if the patient faints either from psychical or cocaine depression. When the patient is thus overcome the reclining position gives immediate relief and allows the operator to proceed with but slight loss of time. The back of the chair should be tipped almost to the horizontal position and the head of the patient supported by a head rest or by an assistant. When the patient is thus reclining the operator should sit by his right side facing the patient. If the operator prefers to stand the patient may be placed upon an operating table or a chair with a high seat.

**Anesthesia** — Cocaine anesthesia is preferable though a general anesthetic may be administered. Flake cocaine is used instead of a solution. A delicate silver cotton wound applicator is moistened in epinephrine solution the excess squeezed from it and then dipped into the cocaine flakes. The loose granules are then gently knocked off and the mucous membrane of the entire septum on both sides is thoroughly massaged or rubbed with it. The membranes should be massaged for about three minutes. After an interval of five minutes they should be massaged again with a fresh preparation. Three applications usually induce complete anesthesia though in rare instances numerous applications are required.

The advantages of this method of applying cocaine over the use of solutions are the speed with which anesthesia is induced and the comparative infrequency of cocaine toxemia. By this method little or no cocaine is swallowed whereas when a solution is used a considerable amount may be swallowed and produce toxic symptoms.

Care should be taken in rubbing the cocaine on the mucous membrane to not traumatize it or produce bleeding as postoperative crusting will likely occur. Cocaine crystals or powder should not be used as the small granules penetrate the mucosa easier than flakes thereby inducing a cocaine toxemia.



A 1 per cent procaine solution should be infiltrated into the columella down to the anterior nasal spine and also beneath the mucoperichondrium adjacent to the columella. The latter infiltration aids in starting the elevation of the mucoperichondrium.

**The Incision**—The choice of the location of the incisions should depend upon the character and location of the septal deviation. If it extends into the vestibule of the nose Hajek's incision should be made at the extreme anterior margin of the cartilage of the septum as shown

in Figure 32 *a*. As the membrane of the vestibular portion of the septum is firmly attached to the fibrocartilage beneath it this incision should only be made when the deflection is far enough forward to render it necessary to remove the anterior portion of the deflected cartilage.

When the deviation does not extend forward into the vestibule Killian's incision (Fig 32 *b*) should be made at the junction of the vestibular membrane with the mucous membrane of the septum as the mucoperichondrium elevates with comparative ease posterior to this point.

The Killian incision is usually preferable and should be made with a sharp pointed knife upon the left side of the septum. Many writers have recommended that it be made upon the side of the convexity of the septum as they believe this allows greater freedom of access in elevating the membrane over the region of convexity. This is ill advised as most operators are more dextrous with their right hand. Furthermore it is unnecessary as the tip of the nose is flexible and may be turned to one side out of the way. Hence it is recommended that the incision be made upon the left side of the

septum except for left handed or ambidextrous surgeons.

The tip of the index finger of the left hand should be introduced into the right nasal chamber to exert counterpressure while the incision is being made. The incision should only extend through the mucous membrane and perichondrium. If it is carried deeper it interferes with the elevation of these tissues.

**The Elevation of the Mucoperichondrium and Periosteum**—This step of the operation is often the beginning of either success or failure in the operation. If the elevation is properly done over the entire area of the deviation on both sides of the septum the subsequent steps are comparatively easy to carry out. If however the elevation is not properly executed and extended over the entire field of the deviation it may interfere with the remaining steps of the operation to such an extent as to defeat its purpose. In the average case in which the cartilage perpendicular plate of the ethmoid and the vomer are involved in the deviation the membrane should be elevated over almost the entire

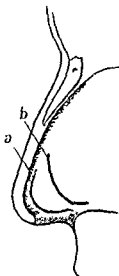


FIG 32—Incisions for the submucous resection of the septum. *a* the Hajek incision. *b* the Killian incision.

area of both sides of the septum. If, however, only the cartilage of the septum is affected, the elevation should be extended about  $\frac{1}{2}$  inch beyond the junction of the cartilage and the perpendicular plate, and down to the floor of the nose. Always elevate at least  $\frac{1}{2}$  inch beyond the area of the tissue to be removed, as otherwise the membrane may be injured in the process of removing the deviated portion of the framework of the septum.

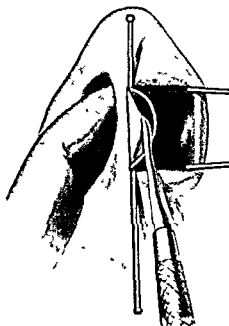


FIG. 33.—The elevation of the mucoperichondrium upon the side of the primary incision in the mucous membrane. The elevation is begun with a sharp or semisharp elevator and is completed with the blunt elevator.

The technic in elevation of the mucoperichondrium may be accomplished in various ways. Some operators prefer small, thin, sharp elevators with which the mucoperichondrium and periosteum are dissected from the framework of the septum. Curved elevators are also used to work around curved portions of the septum. A study of the following descriptive technic will show how the heavy blunt elevators may be used successfully to encompass curved and angular deviations of the cartilage and the perpendicular plate of the ethmoid. The chief reason for using the blunt heavy elevators is the greater speed and the lessened liability of tearing the membrane in the process of elevation.

To start the elevation a sharp or semisharp elevator should be used, care being exercised to get beneath the perichondrium. If the elevator penetrates between the mucous membrane and perichondrium, the surface of the cartilage will present a velvety red appearance as the perichondrium is still covering it. If, however, the elevator penetrates beneath the perichondrium the exposed cartilage presents a glistening

white surface. Great patience is often required to start the elevation properly, this being done the remaining elevation is comparatively easy. The point of least resistance is usually at the upper portion of the Killian incision whereas at the lower portion the perichondrium is often so adherent as to require a knife to separate it from the cartilage.

Having succeeded in starting the elevation abandon the sharp elevator and insert the blunt one (Fig 51) into the small pocket already made. Direct the elevator parallel with the ridge of the nose, as this is the direction of least resistance (Fig 34). Having introduced the elevator almost to the cribriform plate the elevation should be continued backward and downward with the whole length of the shank of the elevator within the pocket of the membrane. The mistake is usually made of attempting to elevate with the tip of the elevator whereas it should be

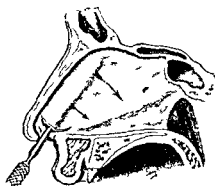


FIG 34 The Harek elevator introduced beneath the mucoperichondrium along the line of least resistance. When thus introduced the elevation should be made with the whole shank of the instrument in a downward and backward direct on to the crest of the vomer. The periosteum along the crest of the vomer should then be incised as shown in Figures 35 to 38.

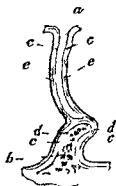


FIG 35 Section through the nasal septum. a quadrilateral cartilage b vomer c agglutination of the perichondrium to the periosteum d periosteum reflected over the crest of the vomer (it is not continuous with the perichondrium) e mucoperichondrium.

done with the shank. With the former it is easy to tear the mucoperichondrium while with the latter the elevation may be rapidly accomplished with but little danger of tearing it. The principle involved is obvious namely a small tip will perforate more readily than a long shank. As a matter of fact the mucoperichondrium and periosteum elevate readily under moderate tension with a broad dull instrument whereas if a small sharp elevator is used extreme care must be constantly exerted to avoid making a perforation.

After introducing the heavy blunt elevator almost to the cribriform plate exert pressure downward and backward with a twisting motion and as a rule, the membrane will strip down to the crest of the vomer in a few seconds or at most in a minute or two. Five minutes or more may be required to start the elevation whereas to complete it will require but a comparatively short time.

The question naturally arises, *How can the elevation be accomplished with the shank of the elevator when the cartilaginous or perpendicular plate portion of the septum is convex?* The operator should remember that these portions of the septum are thin and flexible. Being so, they may be forced with the elevator to the median line and thus temporarily rendered straight. While held in this straightened position the shank of the instrument is passed downward and backward, elevating the membrane as it proceeds. It may also be asked, *How can the elevation be accomplished with the tip of the straight, blunt elevator when there is a perpendicular deviation of the cartilage?*

The procedure is very simple. The tip of the nose is flexible, and the instrument should be held parallel with the anterior portion of the cartilage until it reaches the crest of the perpendicular deviation. The



FIG. 36—Elevation of the membranes of the cartilage and vomer. *a* quadrilateral cartilage, *b*, vomer, *c*, perichondrium, *d* periosteum of vomer with two incisions (*f*) at the crest, *e* mucous membrane, *f*, two incisions through the periosteum along the crest of the vomer, to facilitate the elevation of the membranes anterior to the junction of the perpendicular plate of the ethmoid with the vomer.



FIG. 37—*a* Cartilage, *b* vomer, *c*, perichondrium, *d* periosteum of the vomer, *e* mucous membranes, *f* two incisions through the periosteum along the crest of the vomer. On the concave side the periosteum over the vomer is elevated.

instrument should then be shifted until it is parallel with the cartilage posterior to the crest. The flexibility of the tip of the nose makes this possible, hence a curved elevator is not necessary for the purpose; or the crest may be forced to the concave side, thus rendering it straight and the elevation continued.

The development of the periosteum of the septum throws interesting light upon the technic of the submucous resection of the septum. Histologic examinations of sections of the septum show that the periosteum is not uniformly reflected over the bony portion. Only where bone unites with bone, as where the perpendicular plate of the ethmoid unites with the vomer, is the periosteum continuously spread over the septum. Where the vomer unites with the cartilage of the septum, the periosteum is not continuous with the perichondrium of the cartilage. In the latter

region the periosteum arises from the floor of the nose and passes upward over the lateral surface of the vomer to its crest over which it is reflected and then passes downward over the opposite lateral wall of the vomer to the floor of the nose. The perichondrium is reflected over the periosteum in this region and is closely adherent to it (Figs 35 and 36).

This arrangement of the periosteum and perichondrium explains the well recognized difficulty experienced in elevating the periosteum below the crest of the anterior portion of the vomer when the elevation is begun above it.

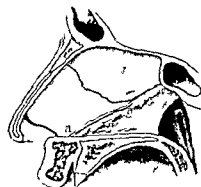


FIG. 38.—Showing the line of incision (a a) through the periosteum along the crest of the vomer to facilitate the elevation of the mucous membranes. A similar incision should be made on the opposite side of the crest.

the nose is the periosteum is continuous from the roof to the floor of the nose.

**The Incision Through the Cartilage**—The incision through the cartilage (after Killian's incision) may be made with a small short bladed sharp knife though it may be done with the tip of a curette or other semisharp instrument. Some operators prefer the latter method believing there is less danger of perforating the opposite mucous membrane. If a knife is used the tip of the finger should be placed in the opposite nostril to exert counterpressure while the incision is being made (Fig 39). The cartilage should be incised very cautiously almost cell by cell with very delicate pressure until the tip of the instrument is felt through the thickness of the opposing mucoperichondrium. Under no circumstance should the opposite mucoperichondrium be incised as this would cause a permanent perforation of the septum unless the incision were immediately closed with sutures. It should be emphasized that if both mucous membranes are perforated at points exactly opposite a permanent perforation will usually follow unless sutured. If the perforations are not opposite a permanent perforation will not result.

If the incision through the cartilage is made with a curette or other semisharp instrument the finger should be placed in the opposite nostril to exert counterpressure while the instrument is being ground through the cartilage. The tip of the finger enables the operator to detect when

The elevation should be begun along the ridge of the nose as shown in Figure 34 and carried down to the upper border of the vomer with the whole length of the elevator. The elevator should then be removed and a short bladed scalpel introduced and an incision made with it along the crest anterior to the perpendicular plate of the ethmoid. The elevator should then be reintroduced and the elevation (on the side of concavity of the septum) continued to the floor of the nose. Posterior to the cartilage the elevation is easily made to the floor of

the entire thickness of the cartilage is penetrated. The cartilage should be incised in a line corresponding with the Killian incision. If, however, the Hajek incision is made the cartilage is not incised, as the incision is anterior to its forward extension. When this incision is made the mucocutaneous membrane is dissected from both sides of the fibrocartilage of the septum with a small, sharp knife.

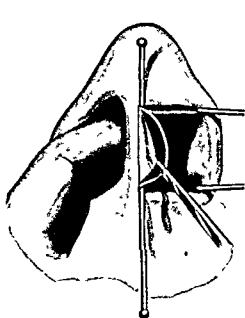


FIG. 39

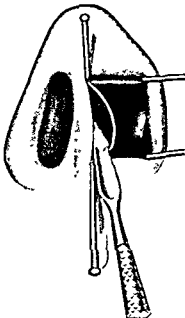


FIG. 40

FIG. 39.—The mucoperichondrium having been elevated the cartilage is incised, care being exercised to avoid perforating the mucoperichondrium upon the opposite side of the septum.

FIG. 40.—The cartilage having been incised the mucoperichondrium of the opposite side of the septum is being elevated. The elevation is begun with a sharp or semisharp elevator, and is completed with a blunt elevator.

**The Elevation of the Opposite Mucoperichondrium and Perosteum.**—When the cartilage is completely incised, the semisharp elevator with its flat surface in apposition with the cartilage, is inserted into the cartilaginous incision. The sharp edge of the tip of the elevator should be moved up and down between the edge of the cartilage and the adherent mucoperichondrium, especially at the upper limit of the incision, as the membrane is less adherent at this point. Having started the elevation the blunt elevator should be introduced and passed upward parallel with the ridge of the nose (direction of least resistance) until its tip is near the cribriform plate of the ethmoid bone. The elevation should then be continued downward and backward, with the shank of the instrument as previously described, and extending over an area considerably larger than the area of cartilage and bone to be removed. Never attempt to elevate below the crest of the vomer when it forms a dense bony ridge, as to do so would only result in an extensive laceration of the membrane.

**The Removal of the Cartilaginous Portion of the Septum**—In nearly all cases this is most easily accomplished with the swivel knife (Figs 43 and 50) though it may be done with Killian's double-edged spoke-shave, a biting forceps, or angular knives. The advantage of the swivel knife is the ease, precision, and rapidity with which it encircles the cartilage, and the further fact that it removes it in one piece, thus allowing the operator to study the specimen as a whole.

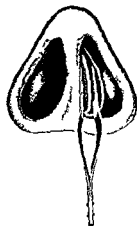


FIG 41 — Showing the Foster septum speculum in position after the membranes are elevated

Before using the swivel knife the mucoperichondria should be distended with a septum speculum to lift them from the cartilage and to provide room for the knife. This exposes the cartilage to full view. The swivel knife may be applied to the cartilage at either the upper or lower portion of the incision. If to the upper portion, the incision will be made upward, backward downward and finally forward along the floor of the nose, thus completely encircling the portion of the cartilage to be removed (Fig 42). If applied at the lower portion of the incision, the cut will extend backward along the crest of the vomer to the junction of the vomer and perpendicular plate of the ethmoid, thence upward and forward, along the antero-inferior margin of the perpendicular plate, and then downward, parallel with the ridge of the nose to the upper limit of the primary incision.

of the cartilage thus encircling the portion of the cartilage to be removed. If the incision is begun at the lower limit of the primary incision it may be necessary first to make a slight cut with a knife or scissors, as the cartilage is often fibrous at this point.

The swivel knife is easily controlled and is an instrument of great precision. The swivel blade follows the direction toward which the tips of the prong are directed. The resistance of the tissues controls the position of the swivel blade so that it always follows the prong tips.

Having encircled the cartilage, it is removed *en masse* with dressing forceps, as shown in Figure 44. Figure 45 shows the perpendicular plate in the depth of the mucoperichondrial pouch after the cartilage is removed.

**The Removal of the Perpendicular Plate of the Ethmoid**—This is accomplished with the Foster-Ballenger bone forceps. They remove a comparatively large piece at each bite and two or three bites remove all that is necessary. The bites may be made without removing the forceps from the mucoperichondrial pouch, a point of considerable importance as each introduction of an instrument into the perichondrial pouch increases the chance of injury to the membranes. The perpendicular plate may also be removed by seizing it with heavy dressing forceps and twisting it from its attachments, though this is a crude and dangerous method, as it may fracture the cribriform plate.



FIG 42

FIG 42 The removal of the quadrilateral cartilage of the septum with the author's swivel knife. The membrane is shown removed to expose the knife to view. In the actual operation the membrane is not removed.

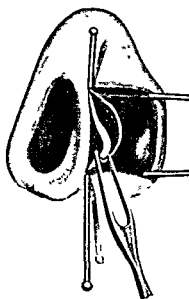


FIG 43

FIG 43 The swivel knife in position at the lower portion of the incision of the cartilage.

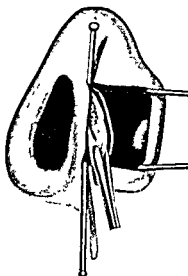


FIG 44

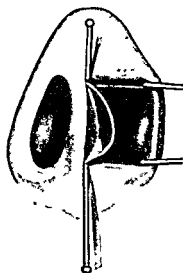


FIG 45

FIG 44 —The cartilage, having been excised submucously with the swivel knife, is removed from the mucoperichondrial pouch with dressing forceps.

FIG 45 —Showing the mucoperichondrial pouch after the removal of the cartilage. The bony crest of the vomer is shown in the bottom of the pouch, while deep in the pouch is shown the perpendicular plate of the ethmoid extending upward from the crest of the vomer. This should be removed with forceps, as shown in Figure 46.



**The Removal of the Vomer**—Various methods are in vogue for the removal of the deviated vomer which often forms the so called ridge of the septum. It is obviously almost impossible to elevate the

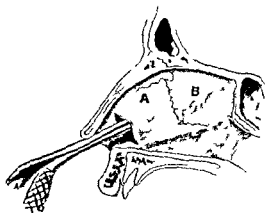


FIG. 46.—The removal of the perpendicular plate of the ethmoid bone with the Foster Ballenger forceps. A the area of cartilage previously removed with the saw and knife. B the area of perpendicular plate removed with the forceps.

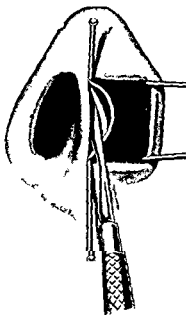


FIG. 47.—The removal of the thickened crest of the vomer with a V-shaped gouge.

mucoperiosteum beneath the crest of the ridge (vomer) as its anterior portion is near the floor of the nose and to attempt to pass the elevator over the margin of the crest would almost certainly tear the tense

mucous membrane along this line. Fortunately it is not necessary to elevate below the crest, as the deviated or thickened bone can be removed without previously elevating the membrane beneath the crest.

An old and approved method of removing the vomer is with Hajek's gouge or some modification of it (Fig. 47). The V-shaped end of the gouge is engaged at the anterior end of the ridge of bone and driven

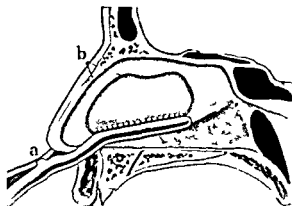


FIG. 48. A method of removing the ridge of bone in the submucous resection of the septum. *a*, the septum forceps grasping the ridge, the blades being external to the mucous membranes. The forceps is rotated on its longitudinal axis as in the Asch operation, thus fracturing the vomer from its lower attachment. *b*, the area of cartilage and perpendicular plate of the ethmoid previously removed; the mucous membrane is shown removed though this is not actually done in the operation.

with a mallet into its substance for a short distance and then the handle of the gouge is depressed and thus partially splinters the bone from its attachment. The gouge is then driven farther into the ridge until it is finally removed in its entirety. As the vomer is loosened it separates from the mucoperiosteum without tearing, provided of course the gouge is always directed parallel with the antero-posterior direction of the crest of the vomer.

Another method of removing the deviated vomer is with a specially devised bone-cutting forceps. Of these I. M. Hurd's is probably the best (Fig. 55). It is powerful, has downward cutting blades and with it the bone may be bitten away with considerable ease.

The vomer should be fractured first from the premaxillary bone at the floor of the nose and then removed with heavy dressing forceps introduced into the mucoperiosteal pouch. During the process of fracture the mucoperiosteum separates from beneath the crest of the vomer and thus allows the long ridge of bone to be removed from the pouch (Fig. 49). In young adults and children this method is not applicable, as the vomer is not yet fully ossified. In adults it is a speedy and an almost painless procedure and results in but little or no shock, as the cartilage and perpendicular plate of the septum have been previously removed.

There is therefore no solid tissue above to communicate the shock to the cranial contents. The technic of the procedure is as follows:

Introduce the blades of the Asch septum forceps into the nasal chambers *outside of the mucoperichondrium* and grasp the deviated vomer firmly, twisting the forceps in its longitudinal axis and fracturing the vomer from its attachment at the floor of the nose. The blades of the forceps should be placed a little above the floor of the nose as they may otherwise tear the mucous membrane at the junction of the vomer with the floor of the nose. The fracture should be thorough in order to

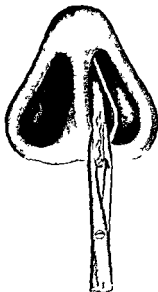


FIG. 41.—The removal of the vomer after it is fractured as shown in Figure 48.

permit the detachment of the fragments from the floor of the nose. Remove the Asch forceps and introduce the tips of heavy dressing forceps into the mucoperichondrial pouch, grasp the vomer and with a tugging-tearing motion lift it from its fractured base. The mucoperiosteum remaining attached below the crest will separate readily and allow the bone to be removed.

#### Inspection of the Field Operated Upon —

After the completion of the various steps of the operation the field operated upon should be subjected to the closest scrutiny. If a portion of the deviated cartilage or bone is left in place it may be found when healing is complete that it will still cause obstruction of the nasal chambers. Every vestige of the deviated framework of the septum should be removed. Bone-cutting forceps of one type or another are usually

used for this purpose in the cartilaginous and perpendicular plate portions of the septum, though the gouge may be more useful for cutting along the floor of the nose. A helpful practice is to insert a finger an inch or two into the nasal chambers as it enables one to detect the presence of bony prominences which might otherwise have escaped notice.

**The Dressing.**—A dressing should be placed in the nasal chambers for two purposes, namely: (a) Coaptation of the membranes and (b) prevention of the formation of a blood clot in the mucoperichondrial pouch.

The dressing most frequently used is  $\frac{1}{2}$  inch self-edged-edge gauze tape soaked in vaseline. Or if desired the Simpson-Berney nasal splint may be used (Fig. 58). The mucoperichondria are first clamped together with the septum speculum, then the gauze or one or two of the splints are introduced into each nasal chamber. If the splints are used the patient's head is then inclined backward and a few drops of distilled water or peroxide of hydrogen are instilled into the ends of the splints.



FIG. 50 — Ballenger's swivel cartilage knife

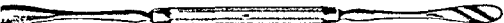


FIG. 51 — Hajek-Ballenger mucoperichondria elevator

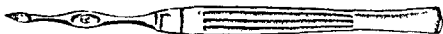


FIG. 52 — Ballenger's mucosa knife

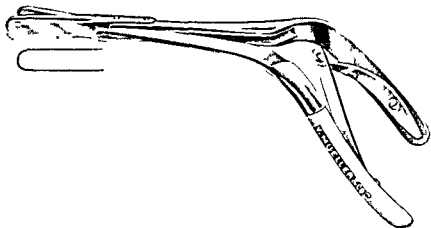


FIG. 53 — Foster-Ballenger perpendicular plate septum forceps

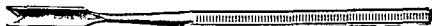


FIG. 54 — Ballenger's septum gouge

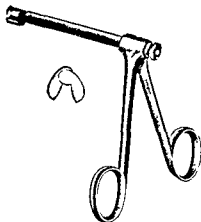


FIG. 55 — Hurd's bone septum forceps

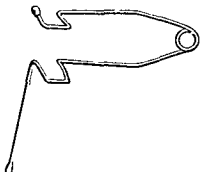


FIG. 56 — Allen's nasal speculum

(Fig 60) This causes them to swell and compress the membranes together. Bruening has devised a wire septum splint which permits drainage.

**The After treatment**—The nasal dressing should be removed in twenty four hours after the operation. Subsequently an alkaline solution or a 1 per cent ephedrine solution may be dropped or sprayed into the nostrils if desired. If crusts form over the incision the patient should be provided with a tube of

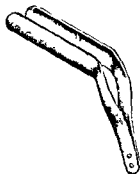


FIG. 57—Ballenger Foster septum speculum



FIG. 58—Simpsom's nasal sponge splint

sterile vaseline and instructed to squeeze some of it into the vestibules of the nose twice a day and to compress the tip of the nose and thus smear it over the mucous membranes. Healing should be completed in from three to ten days, unless one of the membranes has been lacerated in which event it may be somewhat prolonged.



FIG. 59—Bruening's wire septum splint

**Accidents**—This operation is peculiarly liable to certain accidental complications some of which are inherent in the technique while others are the result of the inexperience or temperamental weakness of the operator.

**Incision through Both Mucous Membranes**—The novice is likely to extend the incision through both mucous membranes as the cartilage is easily incised and the most delicate manipulation of the knife is necessary in making the incision through it. Before the operator realizes it the incision has extended through the mucous membrane upon the opposite side. To avoid this accident the cartilage should be incised cell by cell as it were until the point of the knife is perceived by the

tip of the index finger which is in the opposite nostril. Should both mucous membranes be incised along the line of the Killian incision it will be necessary to close one of the incisions with Yankauer's needles or some other method of suture. The sutures should be removed at the expiration of the third day.

**Tears Through Both Mucous Membranes**—Sometimes during the process of elevating the mucous membranes are lacerated at points exactly opposite. Should this accident occur an endeavor should be made to close one of the apertures by Yankauer's method of suturing or to reintroduce the cartilage removed from the septum as suggested and practised by Goldsmith (See Perforation of the Septum).

**Destruction of the Mucous Membrane upon One Side of the Septum** This accident may occur during the elevation of the membrane or during the removal of the cartilaginous and bony portions of the septum with cutting forceps. This is especially true if the elevation of the mucoperiosteum has not been extended over a sufficiently large area. It may also occur while the cutting forceps are in use, the mucous membrane being accidentally engaged in the forceps. This can be avoided by exercising great care before closing the forceps.

**Sinking in of the Ridge of the Nose**—This accident has been reported only a few times and need not be feared except under a few conditions. When it occurs it is due to one of three conditions: (a) The removal of the cartilage too near the ridge of the nose; (b) chondritis following or preceding the operation; and (c) traumatism.

(a) A cartilaginous ridge at least  $\frac{1}{2}$  inch in depth should be left to support the external nose. A greater width is desirable especially if the deviation is traumatic in origin, as in this case chondritis may have weakened the cartilage.

(b) Chondritis or inflammation of the cartilage following the operation may soften the cartilage of the ridge of the nose and cause it to drop or sink in and thus produce external deformity. The nose should be carefully observed for several days after the operation for inflammatory symptoms, and if they occur strenuous efforts should be made to

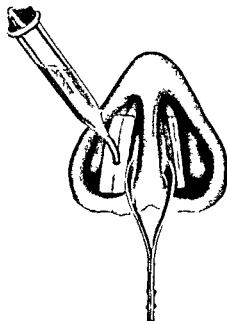


FIG. 60. The Simon sponge-test dressing in position at the close of the submucous operation. The left side shows the tents dry, the right moist and swollen. The Foster speculum holds the membranes in apposition while the tents are being introduced.

combat them. Full doses of one of the sulfonamides usually sulfadiazine should be given and maintained until it is certain the infection has been controlled. Penicillin intramuscularly can be used in place of or with the sulfonamides if the infection is very severe. Irrigations of the nasal cavity with a solution of from 300 to 500 units of penicillin to 1 cc. of physiologic saline is a valuable adjunct to the therapy. Warm fomentations to the nose should also be used.

When the ridge of the nose sinks in after submucous resection of the septum, it is possible to correct the deformity by some form of plastic procedure.

(c) A blow upon the nose after the submucous resection operation might cause a sinking in of the ridge below the nasal bones although it is seldom if ever reported.

**Septal Abscess**—Acute abscess of the nasal septum is not common. It may result from trauma after operations or as a complication of

infectious diseases such as typhoid, influenza, sinus suppuration, small-pox and tuberculosis. Pyogenic bacteria may invade a hematoma thus converting it into an abscess. The symptoms are fever and pain across the bridge of the nose. The nose externally is swollen and red. Anterior rhinoscopy reveals a reddish bulging tumor, occluding both nostrils and boggy to the touch. The treatment of septal abscess involves the use of the sulfonamides and/or penicillin. If pus is present it should be evacuated by incision and drainage.

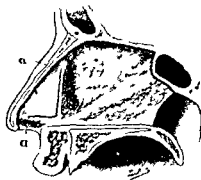


FIG 61. a Freer's incision

**The Freer or Open Method**—According to Freer, his incision is especially adapted to cases in which unusual difficulties necessitate an operative field as open as possible for inspection, as those in which the mucous coverings are very adherent, or in which the operation is performed in the small nostrils of children for deviations with extreme angles or for extensive deep-seated deflections. The open operative field is obtained by means of Freer's reversed L mucous membrane incision (Fig 61), consisting of a vertical limb, made well back in the nose, joined by a horizontal one conducted forward from it along the base of the septum in most cases to the front of the nasal vestibule. These incisions outline a flap which is dissected upward and backward with a suitable blade from its basal line until the vertical incision is reached. The flap is then uplifted by means of the dulled elevator and held forward out of the way by the use of a retractor held by an assistant, these retractors taking the place of a speculum. A large field of cartilage

is thus uncovered in front so that the first incision through it can be made in plain view. It outlines a tongue-shaped flap of cartilage with its base backward and which when uplifted from the mucous coverings of the concave side of the deviation gives a broad entrance into the concavity of the deflection making all of its recesses readily accessible to sight as the denudation progresses so that sharp dissection can be accomplished safely without risk of perforation.

After the posterior portion of the mucous coverings have then been uplifted on the side of the convexity of the deviation the cartilage now entirely denuded is excised with a little keen hook-shaped blade and by sharp elevators. The remains of the cartilage are then detached pos-

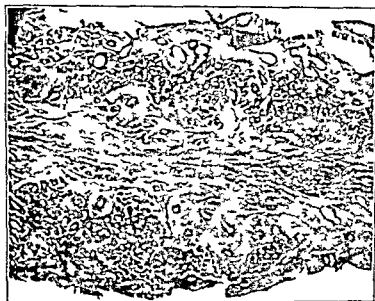


FIG. 6.—Section of septum taken a half year after a submucous resection of bone and cartilage shows no regeneration of either bone or cartilage. It is replaced by a dense fibrous tissue. Age forty-seven years. Specimen kindly loaned by Dr. J. C. Beck.

teriorly from their usual attachments to the side of the vomer by means of long elevators and the bony resection is begun by an incision upon the upper border of the ridge (often hidden) and anterior border of the vomer splitting the periosteal envelop of these structures. The periosteum is then pushed off from their convex and concave sides by means of suitable chisel-edged raspatories and blades and the entire bony deviation bared by them and by the elevators. It is then cut away by the Lister reinforced punch forceps including the ridge of the nasal floor and as much of the vomer and perpendicular plate as is needed. The chisel should only be used in cases in which the ridge is unusually broad.

After the operation the nostril of the side on which the incisions have been made should be packed with narrow strips of lint saturated with bismuth subnitrate and soaked in oil vaseline the strips should be



introduced in layers, in order to avoid injurious bunching and also to hold the flaps in place

Hematoma of the septum does not occur when coaptation of the mucoperiosteal membranes has resulted from the use of suitable dressings



FIG 63 — Sluder's septum operation 1 2 and 3 the lines of incision on

in either method of operating and perforations are rare if the technique is carefully carried out even in extensive bony resections

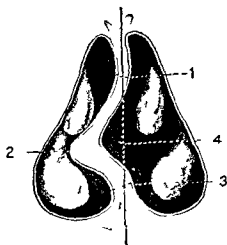


FIG 64

FIG 64 — Sect onal view of the nose before the Sluder operation 1 2 3 the lines of incision as shown in Figure 63 4 the median line of the nose

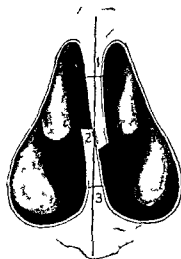


FIG 65

FIG 65 — Sect onal view of the nose after the Sluder operation 1 2 3 the lines of incision as shown in Figure 63 The bands of cartilage overlap and should be held in position with a nasal tube

Authors differ as to the reformation of the cartilage of the septum after its removal According to Beck, no cartilage cells were found

in the tissue after a lapse of two and a half years. The removed cartilage was replaced by dense fibrous tissue. Freer on the other hand claims that the cartilage reforms especially in the younger subjects.

**Sluder's Operation**—Dr. Greenfield Sluder has used a modification of the Watson operation. It has a limited field of usefulness in children with extreme angular cartilaginous deflections.

**Technic**—(a) Local or general anesthesia

(b) Make three parallel incisions through the entire thickness of the septum parallel with the crest (Figs 63 and 64). The middle incision should extend the whole length of the crest. The other incisions are made at the apices of the less acute angles 1 and 2. Two strips of cartilage are thus formed their only attachments being at the anterior and posterior extremities.

(c) Either the upper or lower strip is then forced to the concave side with the index finger or a blunt instrument.

(d) The other strip is likewise displaced to the concave side, thus causing them to overlap as shown in Figure 65.

(e) A Mayer nasal tube is then introduced on the side of convexity to hold the strips in position while union takes place a period of three or four days.

If the opposed surfaces are curetted before coaptation union will take place more rapidly.

#### LATERAL DISPLACEMENT OF LOWER BORDER OF SEPTAL CARTILAGE

The lateral displacement or deviation of the lower or free border of the septal cartilage requires a different surgical procedure for its correction than the classical submucous resection for deviations posterior to this area.

Metzenbaum<sup>1</sup> dissects the mucoperichondrium from the median side only of the deflected portion as far as the angle of deflection. The cartilage is incised at the angle but the incision is not carried through the opposite mucoperichondrium. After freeing the cartilage so it is freely movable it is reset in the mid line. It is frequently necessary to separate or form a groove in the posterior portion of the columella to receive the anterior edge of the replaced septal cartilage. A mattress suture may be necessary to hold the cartilage between the folds of the columella. The primary incision is closed with one or two silk sutures and the nose picked for twenty-four to forty-eight hours.

Peer<sup>2</sup> removes the anterior displaced segment completely but leaves a beam of cartilage along the dorsum of the nose for support. A portion of straight cartilage removed from the posterior portion of the quadralateral cartilage is fitted into a prepared pocket in the posterior edge of the columella and held in place by means of a mattress suture.

Harbert<sup>3</sup> exposes both sides of the deflected portion then makes a

<sup>1</sup> Arch. Otolaryngol. 9: 282 (February) 1909.

<sup>2</sup> Arch. Otolaryngol. 25: 470 (April) 1937.

<sup>3</sup> Arch. Otolaryngol. 31: 341 (February) 1940.

groove in the anterior inferior nasal spine. The cartilage is then placed in position in the groove and maintained by dental wax splints and/or light gauze packing.

In many instances where the tip of the nose is not depressed and adequate support can be left along the dorsum, simple excision of the deflected portion is sufficient. A simple procedure is first to anesthetize the cartilaginous portion of the nasal septum by the local application of cocaine as in the conventional submucous resection, then inject a 1 or 2 per cent solution of procaine hydrochloride beneath the skin of the columella.

An incision is made down to the cartilage over the anterior or free border of the deflected portion. The mucous membrane and perichondrium are then elevated over both sides of the deflected cartilage as far back as necessary for free exposure.

The segment of cartilage protruding into the nasal passage is then separated and removed by means of a small knife or small slender scissors. The portion of cartilage in the mid-line is left for support. If the cartilage is deviated from the mid-line posteriorly to the deflected tip, the cartilage is removed by the technic described for the conventional submucous resection.

If the tip incision does not close easily, it may be united by one or two skin sutures. Frequently packing is all that is necessary.

In some instances additional support to the nasal tip may be thought advisable. In this event a pocket is made in the columella through the original incision, and a segment of cartilage, cut to fit the pocket, is sutured into place by means of a mattress suture.

Nasal packing is placed in one or both nasal cavities to control bleeding if necessary. The packing is removed in twenty-four hours, and the sutures in four or five days.

If the free border of the septal cartilage is associated with a deflected tip of the nose and the latter is to be corrected at the same time, some form of a plastic operation should be done as described elsewhere.

## CHAPTER V

### HYPERPLASIAS OF THE SEPTUM PERFORATIONS OF THE SEPTUM RHINITIS SICC A ANTERIOR

#### HYPERPLASIAS OF THE SEPTUM

Soft hyperplasia (hypertrophies) of the mucous membrane of the septum occur at two points namely (a) At the anterior portion just opposite to or below the inferior margin of the middle turbinate and (b) at the posterior end of the vomer. In the first instance the enlargement closes the anterior end of the olfactory fissure and interferes with the proper ventilation of the superior meatus and the sinuses draining into it. Its reduction is best accomplished as follows:

First induce local anesthesia with a 5 to 10 per cent solution of cocaine applied to the parts with a thin pledget of cotton.

Second make one or two linear incisions through the hyperplastic tissue with the actual cautery at a bright cherry red heat (Fig. 6b).

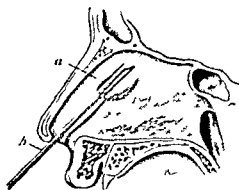


FIG. 6b. The reduction of anterior hyperplasia of the mucous membrane of the septum in the region of the inferior end of the middle turbinate. a linear cauterization of anterior hyperplasia making a small linear incision.

This procedure may be repeated two weeks later if the first application was insufficient to reduce the mass.

In posterior hyperplasia of the septum the same procedure may be followed having first reduced the engorgement of the turbinates with an application of 1 to 1000 solution of epinephrine or 1 to 3 per cent ephedrine.

#### PERFORATION OF THE SEPTUM

**Etiology**—The causes of perforation of the septum may be divided into (a) congenital, (b) chronic granuloma, (c) trauma, (d) acute infection, and (e) atrophic or perforating ulcer.

(i) Congenital perforation is extremely rare. Few cases have been reported.

(ii) Chronic granulomatous diseases, syphilis, tuberculosis and lupus—have caused a considerable percentage of the cases, some authors attributing as high as 50 to 60 per cent to syphilis alone. In the author's experience the percentage due to syphilis is much less than this; syphilis is not however as common in this as in some other countries. Syphilitic perforations almost always include the bony portion of the septum whereas tuberculosis and lupus are limited to the cartilaginous portion. The tuberculous and lupus origin of the perforating ulcer may be determined by finding the tubercle bacilli or tuberculous histologic changes in the tissues. A slow but reliable method of demonstrating the tuberculous process is to inject a guinea pig with some of the tissue from the ulcer. Six weeks later hold a postmortem on the pig and note the presence or absence of a tuberculous process.

(c) Traumatic perforations may include any portion of the septum as they are usually due to surgical procedures though they may be due to accidental violence and to picking the nose with the finger nail.

(d) Acute infectious diseases, as diphtheria, scarlet fever, typhoid fever, phlegmonous abscess, etc., may result in perforations.

(e) Atrophic or perforating ulcer of the septum may occur. Several conditions contribute to the etiology of this type of perforating ulcer. A deviation of the cartilaginous portion of the septum is usually present and on account of its projection into the field of the inspiratory current of air it is subjected to constant mechanical irritation and to the desiccation of the secretions which constantly accumulate upon it. The ciliated columnar epithelium undergoes retrograde changes to a less specialized type of epithelium (pavement epithelium). The dust and other foreign substances in the air also irritate the epithelium and mucous membrane.

The crusts thus formed in this area become adherent and are forcibly blown or picked off with the finger nail, the epithelium coming away with them. Hemorrhagic deposits in the mucous membrane occur and epistaxis is of frequent occurrence. The retrograde process continues until the entire thickness of the septum is destroyed. Infection plays a part in the foregoing process.

**Symptoms**—The symptoms of perforation of the septum vary with the size, cause and location of the perforation. A small anterior perforation sometimes gives rise to a *whistling sound* whereas a large one does not. Crusts if of large size may give rise to the feeling of a foreign body in the nose and if forcibly blown or picked off may cause nasal hemorrhage. Repeated epistaxis should arouse suspicion of a perforating ulcer. Syphilitic ulceration is usually accompanied by an offensive necrotic odor. Many cases will progress to complete perforation without the patient's knowledge of the fact.

**Treatment**—If seen in the ulcerative stage before perforation the progress of the local retrograde changes may be checked by appropriate local cleansing and antiseptic washes and ointments or if due to

syphilis, by the administration of the proper remedies for this disease. When the perforation is complete, little can be done except in a surgical way. Large perforations are not, however, amenable to surgical closure. Small ones may often be closed by proper plastic surgical procedures.

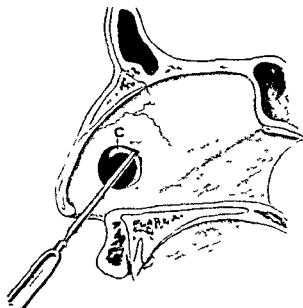


FIG. 67 —The edge of the cartilage around the perforation (C) being removed with the single-tined swivel knife in Goldstein's plastic septum operation.

**Plastic Flap Operation**—Goldstein has suggested and successfully used the following operation. A plastic flap of mucous membrane is turned into the opening and inserted and sutured between the elevated membranes of the two sides of the septum.

*Technic*—(a) Cocaine anesthesia

(b) The rim or edge of the perforation is freshened by pairing off the epithelium and mucous membrane

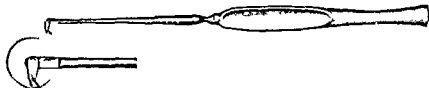


FIG. 68 —Ballenger's mucosa swivel knife

(c) The mucoperichondrium is then elevated for a distance of  $\frac{1}{2}$  inch around the edge of the perforation

(d) A ring of cartilage is then resected for  $\frac{1}{8}$  to  $\frac{1}{4}$  inch from the edge of the perforation, the author's single-tined swivel knife being used for the purpose (Fig. 67)

(c) A mucous membrane flap, the area of which is considerably larger than the perforation, is then dissected from the most convenient surface of the septum and turned into the perforation and tucked between the elevated membranes around the perforation. A trailing swivel knife has been devised for outlining this flap. The method of using it is shown in Figure 69.

(f) When the pedicled flap is in position, three or four Yankauer stitches hold it in position. One surface is covered by epithelium, while the other is left to heal by granulation from the edges of the closed perforation.

**Hazletine's Plastic Operation**—This operation is also only suited to small perforations. It is more simple

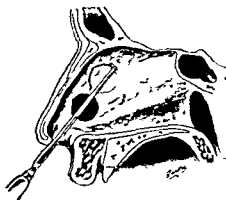


FIG. 69

FIG. 69—Showing the method of outlining the flap with the author's swivel mucosa knife for the closure of a perforation of the septum.

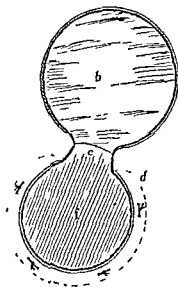


FIG. 70

FIG. 70—f The plastic flap sutured in the perforation. c the pedicle of the plastic flap. b the denuded area from which the plastic flap is removed heals by granulation. d the edge of the plastic flap between the mucoperichondria of the septum.

than the pedicled flap operation, and appears to be a more satisfactory procedure.

**Technic**—(a) Cocaine anesthesia.

(b) Freshen the edges of the perforation and elevate the mucoperichondrium, as in the submucous resection operation.

(c) Make a long curved incision (Fig. 71, b, b) through the mucoperichondrium  $\frac{1}{4}$  to  $\frac{1}{2}$  inch anterior to the perforation, and elevate the ribbon-flap thus made.

(d) Make a long curved incision (e, e) through the mucoperichondrium of the opposite side of the septum,  $\frac{1}{4}$  to  $\frac{1}{2}$  inch posterior to the perforation, and elevate the flap.

(e) Suture the anterior flap to the freshened posterior edge of the mucous membrane of the perforation (Fig. 72), and the posterior flap on the opposite side of the septum to the freshened anterior edge of the

membrane of the perforation as shown in Figure 73. The area *a* and *a'* heal by granulation.



FIG. 71

FIG. 71—Schematic of Haslett's plastic operation for the closure of perforations of the septum. *b b* incision in front of the perforation; *c c* the incision posterior to the perforation on the opposite side of the septum; *e e* the freshened edges of the perforation.

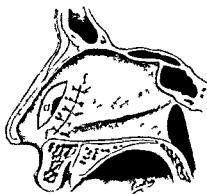


FIG. 72

FIG. 72—Detail of Figure 71 showing the opposite side of the septum; the flap formerly covering area *a* is sutured to the posterior margin of the perforation.

(f) Remove the sutures in twenty-four to thirty-six hours. By this procedure the perforation is covered by two mucous membranes and the lines of suture not being opposite closure of the perforation follows.

**Yankauer's Intranasal Suture**—Sidney Yankauer has devised instruments for intranasal suturing which may be applied in repairing rents in the mucous membrane of the septum following the submucous resection operation in closing the mucous membrane wound of the inferior turbinate after resecting the hyperplastic membrane and bone and in the plastic operations upon the septum for the closure of chronic perforations. The technic is as follows:

**The Introduction of the Suture**—

Very fine catgut suture 18 inches in length should be used. It should be placed in an aqueous solution for a few moments to soften it. The suture may be passed through either flap preferably through the more movable one. It should then be passed through the other flap after first coapting the two flaps. If necessary, the crotch forceps may be used to facilitate the penetration of the flaps with the needle.

**Grasping the Thread**—The eye of the needle should project only  $\frac{1}{4}$  inch through the membranes. One of the threads should then be

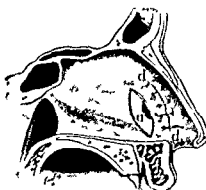


FIG. 73—Detail of Figure 71. *a* The denuded cartilage after the plastic flap (*d*) is sutured.



seized with the hook which may be rotated with the pilot wheel at the end of the instrument until it is in position to seize the thread.

*Withdrawing the Needle*—When the thread is in the grasp of the hook, the needle should be removed from the flaps by rotating it backward until it is free from the membranes. It should then be withdrawn from the nose. The hook should in the meantime be kept close to the needle puncture to prevent the thread from tearing out.

*Withdrawing the Hook*—The hook is withdrawn from the nose with the loop of thread. One side of the loop is then drawn from the nose ready for making the slip-knot.

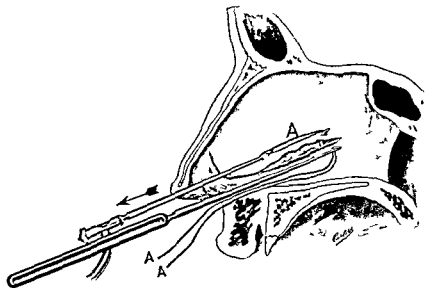


FIG 74.—Yankauer's intranasal suture. 1 A A the suture thread being drawn forward with the hook. The needle is then reversed and withdrawn from the nose, rethreaded and another stitch taken in the torn mucous membrane.

*Making the Slip-knot*—First see that both ends of the thread are outside of the nose and that they are not entangled. To make the slip-knot have one end include half of the thread (9 inches) outside of the nose, the other end being correspondingly shorter. Then make a simple *overhand knot* near the middle of the long ends, and pass the shorter end through the bight of the knot, as shown in Figure 75. Tighten the slip-knot until it binds the through thread. Two threads now come through the knot, one the knot end, the other the slip end.

*Closing the Slip-knot*—The slip knot being drawn tight over the thread it is brought near the nostril. The knot end of the thread is passed through the ring of the suture closer until the ring is near the knot. The end of the thread is then held with the thumb against the handle of the instrument as shown in Figure 76. The left hand holds the slip end, and the ring suture closer is advanced into the nose and the knot closed where the suture passes through the mucous membrane.

The ring passes beyond the point where the suture passes through the membranes and thus makes as firm a knot as may be desired.

The remaining portion of the wound may be closed by making a continuous suture with the longer end of the thread, only using the slip-knot for the last stitch to fix it in place. If preferred for any reason each stitch may be made separately as above described, cutting off the ends as in external suturing.

The sutures should be removed in from two to three days.

*The Safety Knots*—In order to prevent the slip-knot from becoming loose it is advisable to make a true surgical knot, consisting of two overhand knots above the slip-knot.

*Goldsmith's Operation*—When both mucoperichondria are torn during a submucous operation

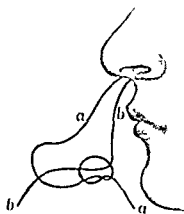


FIG. 75—The slip-knot

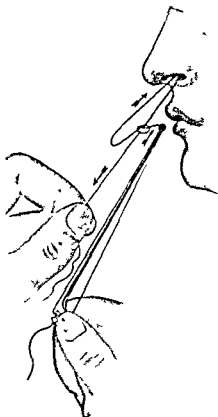


FIG. 76—Yankauer's intranasal suture method of conveying the knot into position in the nasal chamber

thus making it probable that a permanent perforation will follow the cartilage removed may be reintroduced between the membranes, and thus afford a bridge over which the granulating edges of the mucous membranes may extend and close the perforation.

*Technic*—(a) When the cartilage is removed with the swivel knife during the submucous resection operation, it should be placed in normal salt solution to preserve it for use in case the mucous membranes are torn.

(b) If the cartilage is misshapen, it may be straightened or trimmed to adapt it to the requirements of the case.

(c) It should be introduced between the membranes, care being

exercised to bring the torn and ragged edges of the membranes well over the cartilage on both sides

(d) A Simpson-Berney sponge-tent splint should then be introduced into each nasal chamber to hold the cartilage and membranes in position. The sponge-tents should be moistened with sterile water to swell them.

(e) The tampons should be removed on the third day.

By the end of this time the granulations will have extended well over the cartilage and in a few more days will have covered it. In this way the perforation is bridged with new mucous membrane. The cartilage is gradually absorbed, leaving a membranous septum at this point.

Cartilage may also be used to close old perforations. The edges of the perforation should first be pared, the membranes separated around the circumference of the perforation, some cartilage removed, and the foreign cartilage introduced and retained in position as in Goldsmith's procedure for rents and tears during the submucous operation.

### RHINITIS SICCA ANTERIOR

Dry anterior rhinitis, a relatively common disease is atrophic in character and confined to the anterior part of the nasal septum. It is characterized by a dry whitish mucous membrane usually covered by a crust or a thin dry pseudomembrane more or less firmly adherent to the underlying mucosa. The removal of the crusts or membrane may cause an occasional slight bleeding. Constant irritation of the mucous membrane from forceful removal of the crusts or dry secretions perpetuates the trouble and leads to permanent changes in the mucous membrane and perichondrium. The ciliated columnar epithelium is replaced by a flat squamous type with a decrease or absence of glandular function. Superficial ulceration frequently occurs. If extensive or long continued, these ulcers may lead to perforation of the cartilaginous septum.

**Treatment**—Forceful removal of the crusts and dry exudate should be avoided. A bland ointment should be prescribed. If excessive bleeding occurs the bleeding points should be cauterized preferably with the platinum tip electric cautery. Slight bleeding may be controlled by small cotton plugs or by cotton pledgets soaked in epinephrine placed over the bleeding area for a few minutes.

### NASAL SYNECHIA

Adhesions between the nasal septum and turbinates are usually the result of surgical trauma but at times may follow infections of the nasal mucosa in which ulcerative lesions occur. Most nasal synechia are found between the septum and inferior turbinate.

Symptoms, if present, are partial obstruction to breathing on the affected side, and a sensation of a foreign body in the nose.

Treatment by separation of the adhesion and the application of cauterizing agents or ointments to the bases of the adhesions is unsatisfactory as the adhesion usually returns. Insertion of a thin segment of a sheet of dental wax between the turbinate and septum after removal of the synechia will prevent a return of the adhesion in some cases if persisted in for two or three weeks. The sheet of wax should be removed and replaced every two or three days.

Firestone<sup>1</sup> severs the adhesion close to the septum, then excises the adhesion and scar attached to the turbinate with a portion of the mucosa. The area is then curetted down to the turbinate bone forming an elliptoid crater with a vertical axis. The mucous membrane is undermined sufficiently for suturing. The linear suture line reduces the likelihood of a recurrence of the synechia. If the synechia is attached to the middle turbinate it may be necessary to crush the turbinate to obtain sufficient laxity of the mucosa for approximation of the edges.

<sup>1</sup> Arch. Otolaryngol. 31 975 (June) 1910

## CHAPTER VI

### EPISTAXIS RHINOSCLEROMA TUBERCULOSIS PHLEGMONOUS RHINITIS FOREIGN BODIES IN THE NOSE CHRONIC ATROPHIA

#### EPISTAXIS (NASAL HEMORRHAGE)

**Etiology**—Epistaxis or bleeding from the interior of the nose usually occurs from varicose veins (Kiesselbach's plexus) on the anterior inferior portion (Little's area) of the cartilaginous septum but may occur from other portions of the nasal mucosa. The bleeding is not often serious in character though all degrees of severity can occur.

Bleeding from the nose frequently accompanies acute infections of the nose and sinuses. It may follow trauma to the nose intranasal foreign bodies violent exertion high altitude and chemical intoxication as from mercury or phosphorus poisoning. Picking the nose is a common cause.

Many constitutional diseases such as hemophilia purpura syphilis tuberculosis leukemia certain anemias hypertension arteriosclerosis and vitamin C and K deficiency may be characterized by epistaxis. Repeated nose bleeds sometimes accompany rheumatic fever.

Intranasal malignant neoplasms are in almost all instances characterized by the ease with which they bleed on touch. An anterior deviation of the nasal septum predisposes to epistaxis because of its exposed position and the ease with which trauma to the area from picking, etc. may be done.

Occasionally the point of bleeding is higher up and further back on the septum where the source of the hemorrhage is from the ethmoid arteries or veins or telangiectases of other vessels. Usually in these cases the bleeding is profuse and intractable as it originates from an artery in most instances. Severe and persistent bleeding may arise in rare instances from a vessel usually a vein near the nasopalatine or incisive canal. The bleeding point when from this source is found in the inferior anterior portion of the septum. At times severe arterial or venous bleeding may come from the sphenopalatine vessel in the sphenothmoid recess. A postnasal tampon is essential in persistent bleeding from this area.

Persistent bleeding from hypertension frequently originates from beneath the posterior half of the inferior turbinate (Woodruff<sup>1</sup>). The hypertension is usually associated with definite cardiovascular disease. This type of bleeding is most likely to be a sudden gush of blood from the nose or into the nasopharynx. Recurrences are common.

**Treatment.**—The treatment of nasal hemorrhage in most cases is very simple, as a semi-recumbent position combined with the local application of epinephrine or ephedrine readily stop it. In many instances all that is necessary, if bleeding is from the anterior portion of the septum, is to place a small pledget of cotton in the anterior naris and leave in place for a few minutes or an hour or so. Subsequent cauterization with the electric cautery is usually advisable. A blunt-pointed electrode should be used at a cherry-red heat for this purpose, a preliminary application of cocaine is necessary. If blood clots are present, the nose should first be cleared.

Astringent remedies, such as the silver nitrate bead or the chromic acid bead, may be applied to the bleeding area from time to time when the oozing is persistent.

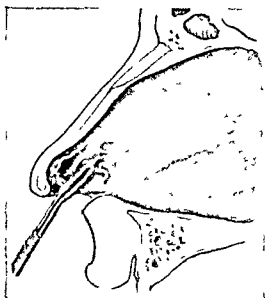


FIG. 77—Cauterizing the varicose veins in Kiesselbach's plexus (Little's area) on the anterior portion of the nasal septum for frequent nose-bleeds.

Gauze tampons in the nose are very effective in persistent bleeding if properly introduced but should not be left in over twenty-four hours. Before introducing the gauze strip it should be saturated in vaseline or oil to prevent adherence to the mucous membrane with the resultant bleeding upon its removal. A  $\frac{1}{4}$  inch selvedge-edge gauze is best. Pack the upper and posterior portion and then work downward and forward until the packing is complete. A Bernay splint tampon, as used in the submucous resection of the septum, may be used if desired if bleeding occurs from the anterior portion of the septum. It absorbs less of the secretions, and is easily introduced and removed without further injury to the mucous membrane.

The administration of calcium lactate gr 5 t i d or the hypodermic injection of fibrinogen or thromboplastin may be of distinct help in reducing the clotting time of persistent bleeders

Morphine is an excellent remedy to induce physical quietness thereby reducing the blood pressure

Bleeding from the sphenoidal artery as it runs under the mucosa on the anterior wall of the sphenoid may be checked by packing a  $\frac{1}{2}$  inch gauze in the region of the sphenoid. Frequently a postnasal pack is necessary

A postnasal pack is most easily accomplished by first tying a small pledget of gauze in the middle of about 2 feet of strong tape. A small size rubber tube or catheter is then inserted through the inferior meatus of the nose (bleeding side) into the pharynx. When the tip of the rubber tube is observed behind the soft palate it is grasped by forceps and pulled out of the mouth far enough to tie one end of the prepared tape to the tube. Friction is then made on the tube or catheter pulling the gauze tampon into the nasopharynx. The strip of tape from the mouth and from the nose are tied together or fastened to the cheek with adhesive tape. The tampon is removed within twenty four hours by loosening the tape and pulling on the end extruding from the mouth.

In rare cases of severe nasal hemorrhage it may be necessary to ligate the external carotid. In selecting the branch of the carotid to be ligated in an intractable nasal hemorrhage it should be remembered that the blood supply of the nose comes from the external carotid artery with the exception of the upper part of the nose which is supplied by the anterior and posterior ethmoid branches of the ophthalmic artery which comes from the internal carotid. However ligation of the internal carotid artery may not control the bleeding because of the various communications of the circle of Willis. It is easier and better to ligate the ethmoid vessels as described for the External Fronto-ethmo-sphenoidal Operation. After a curved medial incision to the inner canthus of the eye the periosteum is elevated to a depth of about  $1\frac{1}{2}$  inches from the bridge of the nose. The anterior vessels are observed in the suture line between the frontal and ethmoid bones along the orbital wall. The posterior vessels are farther back. The anterior and/or the posterior branches are ligated as indicated by the bleeding.

## RHINOPHYMA

Rhinophyma is characterized by a nodular enlargement of the skin of the nose affecting as a rule the inferior and anterior parts in which irregular elevations separated by fissures containing accumulated sebaceous masses are found. Dilated vessels are present on the surface of the nose (Fig 78)

Microscopic sections show greatly increased number and size of the sebaceous glands. The skin and subcutaneous tissue show a chronic inflammatory process.

The disease is thought to follow an acne rosacea

Treatment by roentgen rays and radium have been used with success in some cases. Decortication of the hypertrophic masses by means of a knife or razor is also successful in many instances. The use of a Thiersch graft is unnecessary after decortication.

### SCLEROMA, RHINOSCLEROMA

**Definition.**—Scleroma is characterized by a cartilage-like hardness and nodular enlargement of the nose and other portions of the upper

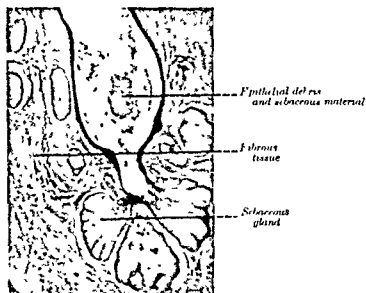


FIG. 78.—Rhinoscleroma of the nose showing a fissure with numerous large sebaceous glands at its base ( $\times 15$ )

air passages. The affected tissues have no tendency to ulceration or to inflammatory reaction either in the growth or in the contiguous parts. Rhinoscleroma frequently affects the other divisions of the respiratory tract.

**Etiology.**—Scleroma is considered by most writers to be infectious. There has been some difference of opinion as to whether the organism described by von Frisch and called the *Bacillus rhinoscleromatis* is the causative factor. The work of Figi and Thompson<sup>1</sup> tends to confirm this view. Scleroma is chiefly confined to Austria and southwestern Europe. Some cases have been reported in America, but a large majority of these were born in Poland and Austria. It usually begins in youth, and most cases are observed between the ages of fourteen and forty-five. Sex and heredity seem to have no influence, though there is apparently a family predisposition to the disease probably due to its contagious nature.

<sup>1</sup> Jour. Am. Med. Assn., 91, 637 (September), 1928.



**Bacteriology**—The hard cartilage like nodules may affect the skin and the mucous membrane of the nose pharynx larynx and trachea. They spread with greater freedom in the mucosa than in the skin. The hard nodular masses or plaques contain the encapsulated bacillus of

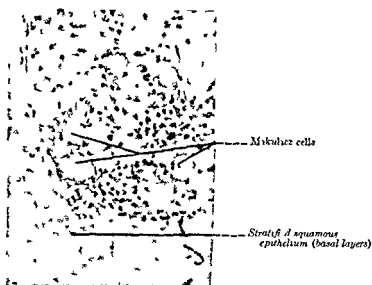


FIG. 9.—Scleroma of the nose (X 400)

rhinoscleroma which is similar to Friedlander's bacillus though the latter is not always encapsulated. The bacillus of rhinoscleroma is more rod-shaped and stains by Gram's method is motile non spore bearing and aerobic. It always has a capsule in culture as well as in the tissues. It occurs singly and in pairs. Gelatin plates show yellowish white granular bodies in two or three days. In gelatin tubes the growth appears along the needle track as a whitish granular line with an almost hemispherical elevation on the surface. The growth in the tube has the appearance of a round headed nail. When grown upon agar it appears as a dirty whitish moist layer on either side of the needle track. On potato the growth is creamy white. It grows rather rapidly at a temperature of  $37^{\circ}\text{C}$ . It is pathogenic for mice guinea pigs and rabbits.

**Pathology**—The histologic changes are inflammatory in character and usually begin on the nasal septum trachea or larynx. In rare instances the reverse course is pursued. The skin and mucous membrane of the nose assume a smooth nodular appearance of cartilage like consistency which pits little if at all upon probe pressure. They have been likened in external appearance to a keloid. The parts are sensitive to the touch but are otherwise free from pain. The substance of the

swelling is composed of large plasma cells irregularly distributed in all layers of the mucous membrane and in the submucous tissue. They accompany the blood vessels in the new portions of the growth. The plasma cells do not contribute directly to the hypertrophy but it is possible that they become changed partly into spindle cells and then give rise to the formation of new fibrillary tissue. Two forms of retrograde metamorphosis occur in the plasma cells. These may be transformed into swollen hydropic so-called Mikulicz cells or into hyaline degenerated cells probably identical with the so-called Russell's fuscinophiles described under Colloid Degeneration. The hydropic cells lie close together have a distinct contour and spongy cytoplasm dilated into large masses in which there is a smaller mass within a faceted nucleus. In this stage one often sees from six to

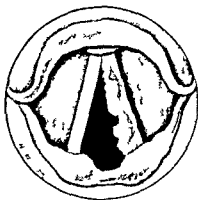


FIG. 80 — Scleroma of the larynx affecting the right cord

eight bacilli in the cells near the nucleus which lie always at regular distances. This stage appears however to be rapidly finished and when the cell membrane breaks the fluid contents together with some of the bacilli find an exit and fill some of the nearest lymph spaces. These cells are however intimately related to the direct action of the bacilli.

**Symptoms** — The changes in the external appearance of the nose while presenting many of the characteristics of keloid are nevertheless rather easily differentiated from it by the whole symptom complex. The tissue at the tip of the nose becomes infiltrated, hard and nodular. The nose broadens and becomes firmly fixed to the face. The tissues become more and more thickened until the breathing is more or less occluded. The color of the skin varies from a red to a bluish or brownish red. The skin is traversed by small blood vessels and is usually shiny though it may be finely wrinkled. The extension of the growth is rather slow, requiring several months to reach the nasopharynx. The infiltration often interferes with the movements of the lips, fauces and larynx but very rarely with that of the eyes and ears. There is no tendency to ulceration and discharge or to edema and inflammation of contiguous parts.

Laryngeal stenosis may give rise to serious or even fatal dyspnea otherwise the disease does not materially affect the general health.

**Diagnosis** — Rhinoscleroma should be differentiated from syphilis, tuberculosis, leprosy, epithelioma and keloid. The disease is exceedingly rare in this country, hence it is natural to infer that a suspected case in a native born American is probably not rhinoscleroma but that it is either syphilis, epithelioma or keloid. Rhinoscleroma presents a hard

nodular growth which usually begins at the anterior end of the nose and spreads gradually to the deeper recesses of the respiratory tract without pain but with some tenderness upon pressure and without tendency to ulceration or inflammation of the surrounding tissues. In syphilis there is inflammation while in epithelioma there is pain ulceration and discharge. In keloid the similarity is often so striking that it may be necessary to demonstrate the absence or presence of the germ of rhinoscleroma in order to make a differential diagnosis.

**Treatment** — Thus far the extirpation of the diseased tissue has been tried with negative results as to the cure of the disease. The surgical extirpation of the diseased tissue has almost invariably been followed by recurrence. Ircheotomy should be performed when suffocation is imminent.

Thiosinamin apparently softens the tissue (Glass) as it does in keloid it may therefore be of some therapeutic value. Iridenthal suggests the injection of Coley's fluid as in sarcoma. The iodids arsenic antimony and mercury have been tried with but little success. Vaccines have given little results. The cautery has not been successful.

The treatment with roentgen rays and radium is more striking as the lesions clear and the induration disappears rapidly. Obstructive bands and adhesions may be removed surgically before instituting treatment with the roentgen rays. Actinotherapy may be combined with the roentgen radiation.

## FURUNCULOSIS OF THE NOSE

**Definition** — Furunculosis of the nose is a superficial abscess formation which may occur in any part of the nose and does not differ materially from the same process in the other parts of the body.

**Etiology** — The abscess is usually located in the sebaceous glands and hair follicles of the nasal vestibule, i. e. that portion covered by the vestibular skin. In addition to the usual causes of furuncles elsewhere there is frequently a history of injury as from picking the nose or a recent acute rhinitis. One or more furuncles may be present at a time or they may occur in quick succession. The hair follicles of the vestibule offer favorable sites for the infection. Recurrence most commonly takes place in diabetics or in those in whom an impoverished state of the blood exists. A staphylococcus is the usual organism recovered.

**Symptoms** — There is more or less throbbing pain swelling redness and tenderness. Elevated areas characteristic of boils may be seen upon inspection. When they are well advanced the center of the elevation is yellowish from the contained pus. The pain is often intense on account of the closely attached and unyielding nature of the tissue composing the parts.

The boil in the nose may suddenly terminate fatally by extension of the infection through the venous channels to the cavernous sinus due either to attempts at opening usually with a dirty needle at home or by squeezing rupturing Nature's barrier driving the infection into the less resistant subcutaneous tissue or picking the head off the furuncle with a dirty finger nail or by too free incision opening up new avenues

outside Nature's barrier. In other cases it seems to travel through the venous channels by reason of lack of resistance to infection.

Swelling of the lids, chemosis, pupillary changes, deep-seated head ache, beginning ptosis and exophthalmos, accompanied by lacerimation, congested retinal veins and symptoms of sepsis are indicative of a cavernous sinus thrombosis when secondary to a furunculosis of the nose or upper lip.

**Treatment** — Manipulation or squeezing of the boil should be avoided due to the danger of forcing the infection into the blood channels.

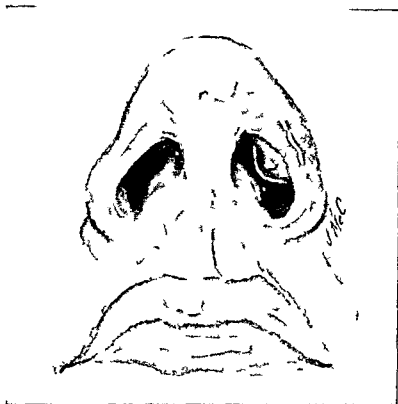


Fig. 81. Frontal view of the lateral margin of the left na-

se. Heat in the form of hot applications give relief from pain and hasten the suppurative process. Soak all pledgets of gauze or cotton soaked in a saturated solution of hot boric acid and place in the nasal vestibule and over the tip of the nose is an efficient way of applying the applications.

If seen early before pus formation the application of sulerythema doses of roentgen ray will often abort the process. If pus has formed the abscess may be incised within the limits of the abscess walls care being taken never to go outside of Nature's barrier. Pressure or suction if applied at all should be done very cautiously. A staphylococcus bacteriophage has been reported upon favorably in some cases.

If there is evidence of an extension of the infection into the blood or

lymph channels or into the tissue space. Full doses of the sulfonamides and penicillin should be given.

### FOREIGN BODIES IN THE NOSE

Foreign bodies in the nose may be animate or inanimate.

*Screw worms* in the nose have been reported by a number of writers especially in the warmer sections.

The screw worm fly is attracted by a foul smelling discharge from the nose or the ear and it need be in the nose but for a moment in order to deposit its eggs.

The symptoms are those of an acute sinusitis that is a profuse unilateral mucopurulent discharge usually with an offensive odor nasal stenosis usually complete on the affected side pain or headache in some form and bleeding or a bloody mucopurulent discharge in most instances. The worms cling with great tenacity to the tissues. Much destruction of tissue with bulging of the walls may occur in the more severe cases.

The diagnosis is made from finding the eggs or worms within the secretions or cavities of the nose or sinuses.

Chloroform is the most effective remedy and may be administered by inhalation or in diluted solution with a syringe. Calomel fumes are also of value but do not act as quickly as chloroform.

*Inanimate foreign bodies* include almost every kind of inert substance small enough to be introduced into the nose and some that are too large to be introduced through the nasal opening such as from injuries gunshot wounds etc.

In most instances the foreign body is voluntarily introduced by the patient. Young children have an inordinate desire to introduce such substances as beans peas beads etc into their noses. Seeds such as beans peas etc if in the nose long enough may sprout or become greatly softened.

The removal of the foreign body may be accomplished through the interior nasal opening without the use of a general anesthetic though in some cases this may be necessary. Forceps with good grasping tips may be used to seize a rough or easily grasped object but a curved probe is better adapted to remove such objects as beads or marbles. The curved portion of the probe should be passed behind the object and pulled forward and thus the object readily comes out. Some bleeding may result.

### RHINOLITHS

**Synonyms** — Nasal concretions nasal calculi

These formations are rare in the nasal spaces. They usually have a foreign body nucleus of bacteria blood pus cells mucus crusts or some foreign body from without the body. They are usually unilateral and located in the majority of instances in the lower portion of the nasal cavity. They vary in size from small granules to an ounce or more in weight. The condition is found more commonly in adults than in children and more often in the female than the male. They may have a gray

brown or a greenish black color. They may be soft and crumbly or hard and brittle. They are largely composed of calcium and magnesium salts principally carbonate and phosphate with traces of sodium chlorid. The stones vary in shape but usually conform to the shape of the nasal cavities.

The presence of some nasal disease or deformity which produces varying degrees of nasal obstruction resulting in poor drainage of the nasal cavity favoring stasis of the secretions is an important factor in the formation of the true type of rhinolith. The inorganic salts adhere to the nucleus and as it enlarges it has a tendency to fill the nasal space impinging itself in the surrounding tissues.

The symptoms of rhinolithiasis vary from a slight one-sided nasal discharge or obstruction to marked structural changes. A unilateral fetid discharge, hemorrhages or obstruction to breathing are symptoms usually observed.

Complications are not unusual. There may be pressure atrophy of the adjacent structures and subsequent decomposition, deviation of the septum to the opposite side and also destruction of the septum in rare instances. More rarely perforation of the palate and facial paralysis have been noted. The involvement of the sinuses on the affected side is commonly observed in most reported cases.

In the differential diagnosis syphilitic sequestration, calcified polyps, osteomata, necrotic bone and malignancy must be considered.

The treatment is removal of the calculus. This may be done under a local or a general anesthetic.

If the calculus is large it may be necessary to break the calculus into bits in order to facilitate its removal.

### ATRESIA OF THE POSTERIOR CHOANÆ

Choanal atresia is a condition consisting of a closure of one or both posterior nares. The occlusion may be membranous or bony or a combination, unilateral or bilateral, complete or incomplete. It may be acquired or congenital.

**Etiology**—The acquired type is more apt to be located in the pharyngeal region. It may be the result of some severe infection near the posterior nares which results in subsequent formation of a cicatrix. These infections are syphilis, diphtheria, tuberculosis and from trauma.

The congenital type of atresia is located in the posterior choanæ. Various theories of the causation have been mentioned such as (1) persistence of the naso-buccal membrane, (2) persistence of the buccopharyngeal membrane (Stewart), (3) a median overgrowth of the vertical and horizontal processes of the palatal bone and (4) secondary to an intra uterine inflammatory condition. Persistence of the naso-buccal membrane is probably the most common cause of the congenital type.

**Pathology**—In congenital choanal atresia the thickness varies from a membrane up to 12 mm. The occluded wall is usually situated even with or just anterior to the posterior end of the vomer. In adults unilateral occlusion is far more frequent than bilateral and bony occlusion

is far more common than the other types. In infants the bilateral occlusion is probably of more frequent occurrence.

The mucous membrane and turbinates may after some years show atrophy or polypoid degeneration.

**Symptoms**—The symptoms of bilateral complete occlusion are absence of breathing through the nose, difficulty in nursing in very young infants, in fact, it may be necessary to feed the infant by means of a spoon or a stomach tube. If the occlusion is partial the condition may not be recognized.

The nasal cavities are filled with a secretion of a peculiarly glairy gelatinous consistency, which irritates the lips and nostrils. Anosmia always accompanies this condition.

Examination of the posterior nares by means of a naso-pharyngoscope inserted through the mouth or the palpating finger in the nasopharynx may aid in the diagnosis. It is impossible to pass an instrument through the nose.

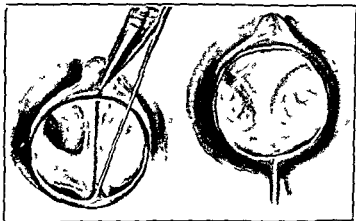


FIG. 82.—A. Nasopharyngeal mirror exposure of a unilateral choanal atresia. B. Nasopharyngeal mirror exposure of a bilateral choanal atresia. (Kazanjian and Otol. Rhinol. Laryngol.)

**Treatment**—The treatment of atresia of the posterior choanæ is the surgical removal of the obstruction. The technic employed would depend upon the type of obstruction, whether membranous or bony, the age of the patient and the presence or absence of any associated pathologic condition.

A membranous occlusion may be removed surgically or may be perforated with a probe or other instrument and the opening enlarged by a series of dilatations with good results in many instances (Lemere<sup>1</sup>).

Destruction of the membranous type by means of electrocoagulation has been used successfully (Morgenstern<sup>2</sup>). In infants and children

chloroform is used for anesthesia. A small electrode, insulated except at the tip, is inserted through the nostril until contact is made with the membranous occlusion. From 300 to 350 milliamperes of current (Bourgeois and Laroux<sup>1</sup>) are used. Four or five treatments from ten days to three weeks apart may be necessary. The rubber covered finger in the nasopharynx acts as a guide to the electrode. This method is not suitable for osseous obstructions.

In bony occlusions which constitute about 90 per cent of the congenital cases (Schwartz and Isaacs<sup>2</sup>) it is necessary to perforate and break down the partition by means of chisel, drills, curettes, trephines, etc. The entire bony obstruction must be removed as well as the posterior portion of the nasal septum. This latter procedure is necessary to avoid a later cicatricial closure. In removing the occlusion care should be taken to avoid injuring the pterygopalatine canal which is situated above and just anterior to the choanal orifice, or the posterior palatine canal situated just behind the choanal opening on the outer wall.

Various approaches to the obstructing wall have been tried. Von Fick<sup>3</sup> in 1911 and White<sup>4</sup> in 1915 removed the posterior end of the vomer and bony occlusion by way of a submucous resection of the septum. This method would be feasible for adults only. Ruddy<sup>5</sup> suggests a transpalatine approach for infants and small children and reports a successful result. Kazanjian<sup>6</sup> in his adult patients incises the base of the columella and extends a through and through incision along the base of the septum backward to the anterior tip of the vomer. The incision is then carried upward along the junction of the quadrilateral cartilage and perpendicular plate of the ethmoid thereby creating a movable flap of the cartilaginous septum. The vomer is then removed back to the atresia creating a large opening through which the occluding bone is accessible for removal. The cartilaginous septum is then sutured into place. A large posterior nasopharyngeal space is thus created.

Prevention of cicatrization with subsequent closure has received much attention. Various methods have been mentioned and tried such as nasal tampons, metal and rubber tubes or catheters, postoperative electrocoagulation and skin grafting of the exposed areas. All have been successful in selected cases. If complete removal of the obstructing tissue including a portion of the posterior edge of the nasal septum is done subsequent closure will probably not occur if excessive granulations are prevented.

### ATRESIA OR STENOSIS OF THE NASOPHARYNX

**Etiology**—Stenosis of the nasopharynx is usually acquired. In acquired stenosis syphilis has been thought to be the most common cause.

<sup>1</sup> Rev. de laryngol. 43: 883 (November) 1922.

<sup>2</sup> Arch. Otolaryngol. 35: 603 (April) 1947.

<sup>3</sup> Laryngoscope 28: 571 1918.

<sup>4</sup> Arch. Otolaryngol. 41: 432 (June) 1945.

<sup>5</sup> Ann. Otol. Rhinol. and Laryngol. 51: 704 (September) 1942.



Trauma diphtheria tuberculosis congenital anomalies and inflammatory lesions may be etiologic factors. Trauma incident to the removal of tonsils and adenoids is a direct cause of stenosis in many cases. The various granulomatous tumors may be factors in others. The swallowing of caustics may produce the stenosis in some instances.

**Pathology**—The soft palate and posterior pillars are adherent to the posterior pharyngeal wall leaving as a rule a small central opening connecting the nasopharynx and pharynx.

**Symptoms**—The symptoms are dependent on the degree of obstruction. If present they consist of partial or complete nasal obstruction with blocked drainage. Changes in the nasal mucous membrane with sinusitis or deafness may follow. The sense of smell is interfered with as a rule. The voice has a nasal quality. The continued mouth breathing may produce changes or irritation of the pharynx and the lower respiratory tract.

**Treatment**—Many procedures have been advocated for relief from cicatricial stenosis of the nasopharynx such as incision and subsequent dilation or incision and later cauterization. These measures have almost invariably failed.

Dierthermy using a specially constructed knife has been used for destroying the scar with some success.

Various plastic operations in which flaps of mucous membrane from the cheeks soft palate etc. have been used with little success. Skin flaps introduced through a subhyoid pharyngotomy opening have also been suggested.

Mackenty cuts flaps in the soft palate and by means of sutures doubles the flaps upward and backward thus applying the two raw surfaces against one another. Dilatation is practised afterward. In the event of failure or inability to secure satisfactory flaps from the posterior pharyngeal wall he produces a cleft in the soft palate.

**Technic**—A horizontal incision is made along the normal palatal curve from one pillar to the other. From the center of this a perpendicular incision is carried through the soft palate upward well beyond the upper line of the atresia. The adhesions are freed laterally to this on both sides. The soft palate is now divided into two triangular portions. The anterior and posterior mucous membranes of these flaps are united by a continuous or interrupted stitch across their raw edges. To make this more effective and to do away with tension as far as possible a V shaped trough is made in the edges of the flaps a strip of tissue being removed from the two sides of each triangle. This requires a sharp knife and an assistant to keep the tissues taut.

Dilatation should be carried out after both of these operations to such an extent or for such time as seems indicated.

Gooden<sup>1</sup> suggests a method for maintaining an opening in the incised scar tissue. A No. 16 French catheter is passed through the nose and into the pharynx. The end of the catheter in the pharynx is

then brought back through the opposite posterior nasal opening and through the nose. The catheter is cut at a proper length to make a loop extending below the opening in the pharynx. The two ends extending from the nose are tied together in front of the columella. The patient is instructed to move the catheter loop back and forth several times a day thus preventing adhesions until healing occurs. The tube is worn from four to six weeks or longer if necessary.

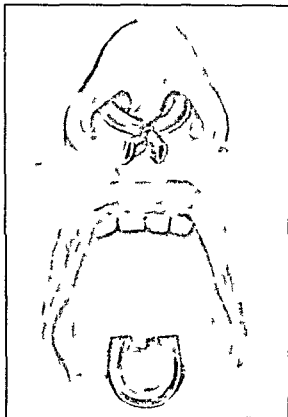


Fig. 83 Rubber tubing in place (Goodyear) for nasopharyngeal atresia

**Nichols Operation** — Nichols inserts a seton at the base of the web until cicatrization has taken place. He then incises to this point and inserts a silk suture through the nasopharyngeal opening into the lateral region of scarring. The suture is tied and left in place until a cicatrized tract develops. The posterior border of the soft palate is then freed to this point.

Fig<sup>1</sup> uses the same procedure but adds a small lead weight clamped over the ends of the suture which is permitted to cut through by itself. This usually requires from one to two weeks. After the suture cuts through another suture taking a wider lateral bite is inserted. Sub-

sequent dilation is employed as a rule. The treatments require from a few weeks to several months. The silk suture method seems to give much better results than the plastic procedures.

### NASAL TEETH

Teeth growing upward into the nose have been observed a number of times.

**Etiology** — The cause is unknown. An obstruction to the downward growth of the teeth is thought to be a factor. Supernumerary teeth are present in the majority of cases. A single tooth usually the maxillary first incisor or canine is involved but multiple nasal teeth may be seen at times.

**Symptoms** — Symptoms if present are possible obstruction to breathing on the affected side, crusting and ulceration, nasal discharge frequently with odor, bleeding from the region of the tooth (Hitschler<sup>1</sup>) and neuralgic pain simulating trifacial neuralgia.

The diagnosis is made from the examination and the roentgen ray.

**Treatment** — The treatment if indicated is by surgical removal.

### ATRESIA OF THE ANTERIOR NARES

Atresia of the anterior nares is rare. It is much less common than atresia of the posterior nares. It may be congenital or acquired, unilateral or bilateral, complete or partial.

**Etiology** — Acquired atresia may be due to trauma, infections, caustics or faulty surgical procedures.

The congenital form has been attributed to inflammatory processes during intra uterine life (Cheate<sup>1</sup>), a developmental anomaly of the pouch of Jacobson's organ (Williams), marked proliferation of the epithelium (Schaeffer), persistence or plugging of the anterior nares during early embryonic life or according to Thomson the condition may be caused by a web of skin at the junction of the vestibule and the nasal chamber (Cinelli<sup>2</sup>). Jervet<sup>3</sup> suggests the condition may be the result of faulty dissolution of the tissues before birth, somewhat similar to the web found at times in the lower end of the nasolacrimal duct in the new born.

**Symptoms** — The symptoms of complete bilateral occlusion in the infant would be difficulty in nursing, mouth breathing and possibly asphyxia at times.

**Treatment** — The treatment consists in the surgical removal of the atretic membrane and at times the use of Thiersch grafts to line the raw area to prevent granulations and contraction. Before applying the skin graft the field must be dry and the graft must adhere snugly to the exposed area. The graft is held in place for ten days or more by means of a rubber tube or light packing.

<sup>1</sup> Arch. Otolaryngol. 28: 911 (December) 1938.

<sup>2</sup> Ann. Otol., Rhinol. and Laryngol. 49: 912 (December) 1940.

<sup>3</sup> Ann. Otol., Rhinol. and Laryngol. 53: 182 (March) 1944.

## CHAPTER VII

### ALLERGY HAY FEVER HYPERESTHETIC RHINITIS ASTHMA NASAL HYDRORRHOEA

#### ALLERGY

**ALLERGY** or hyper-sensitiveness to many usually harmless substances commonly encountered in the diet or environment manifests itself in such diverse conditions as hay fever hyperesthetic rhinitis asthma eczema, urticaria and migraine.

The term allergy may be taken to mean a pathologic hypersensitivity of body tissues to a substance (usually protein) which is harmless to the majority of individuals.

**Etiology** — At least two factors are involved (1) a certain constitutional sensitization usually inherited and (2) the presence of the specific exciting substance. The underlying mechanism of this hypersensitivity is not known.

The most commonly accepted theory of the mechanism of allergy is the 'cellular theory'. The cellular theory of allergy is explained by the exciting agent or allergen coming in contact or uniting with the antibody in the sensitized cells producing a colloidal shock reaction with the production of histamine which may be a factor in the production of allergy or produce effects similar to the reactions which occur in allergic states.

The 'humoral theory' of allergy is explained by the antibody in the body fluids or cells coming in contact with the exciting agent or allergen in the blood with the production of a toxin (anaphylatoxin) which produces the lesions and symptoms.

Protein or protein derivative substances such as pollen moulds, carbohydrates drugs and even metals or physical agents can lead to these reactions.

Heredity is an important factor in allergy. The nature of this influence is not known but it seems to be a dominant Mendelian character. It has been estimated that from 5 to 10 per cent of all people give some clinical manifestations of allergy. An even larger number will give positive skin tests without clinical evidence. Rowe<sup>1</sup> in an analysis of 2000 students at the University of California found a personal history of allergy in 33 per cent and a family history of allergy in 35 per cent. Vaughan<sup>2</sup> including migraine, gastro-intestinal allergy and food idiosyncrasies with urticaria, eczema hay fever and asthma found evidence of personal allergy in 60 per cent of the total population in a community of over 500 persons. Women are more likely to transmit the condition than men.

Contributory factors are reflex irritation and foci of infections, especially in the production of asthmatic attacks.

<sup>1</sup> Arch Otolaryngol 21 653 (June) 1935

<sup>2</sup> Jour Allergy 5 184 1934

The various agents may gain access to the body by inhalation ingestion contact or injection

**Pathology** — The allergic nasal and sinus mucous membranes show an edematous infiltration of the subepithelial stroma with hypersecretion and desquamation accompanied by a cellular infiltration in the tunica propria of eosinophiles lymphocytes and plasma cells. Large numbers of pus cells in the secretions without eosinophiles usually indicate a purely suppurative process. This suppurative process is a secondary manifestation in most cases of nasal allergy especially in the latter part of the season in hay fever.

In chronic allergy the mucous membrane will also show the characteristic edema but with an accompanying fibrosis. A preponderance of eosinophiles accompanied by lymphocytes and plasma cells will also be found. Hyperplasia of the mucous membrane is a later manifestation.

The most constant change of the bronchial mucosa in asthma is an edema or serous infiltration of the subepithelial connective tissue accompanied by an eosinophilic infiltration with the addition of small lymphocytes and plasma cells. Some connective-tissue hyperplasia is present around the arterioles and in the tunica propria. The surface epithelium may be thickened from many layers of stratified columnar cells. A loss of cilia and areas of desquamation may be observed in some cases.

The glandular structures usually are dilated and filled with secretion. In advanced stages an atrophy of the glands may occur. Variations are found in different cases or in the same specimen.

Bone changes if present are confined as a rule to the ethmoid cells where a tendency to decalcification may be seen.

The differential cell count taken from the edematous areas may show eosinophiles 20 to 40 per cent lymphocytes 25 to 45 per cent and plasma cells 15 to 30 per cent. The number of eosinophiles in the blood the tissues and in the nasal secretions shows variations in the same specimen or in different cases. Eosinophilia is also found in worm infections myelogenous leukemia and at times in scarlet fever and some skin diseases.

The epithelium in vernal conjunctivitis is swollen and a connective-tissue proliferation with sclerosis and hyalinization is present. The glands and secretions are full of eosinophiles and lymphocytes.

Nasal mucous polyps are common in allergic conditions of the respiratory tract and rare in patients with non allergic diseases. A personal or a family history of allergy or positive skin tests can be obtained in most cases.

**Examination of the Nose** — The examination of the allergic nose reveals the mucous membrane discolored grayish pink or bluish gray with swelling or edema particularly of the inferior turbinate and in the middle meatus or anterior ethmoid regions. In more advanced cases hyperplasia and polypoid formation may be observed. The polypi are attached usually in the anterior and posterior ethmoid regions. The sinuses particularly the ethmoid cells and the maxillary antrums may show edema hyperplasia and polypoid degeneration.

Vernal conjunctivitis is characterized by itching photophobia and lacrimation. Mucus is usually present, especially in the morning. The secretion contains eosinophiles as a rule.

**Differential Diagnosis**—The differential diagnosis should be made from sinusitis, reflex nasal neurosis, acute rhinitis, mechanical irritations and cerebrospinal rhinorrhea. In these non-allergic manifestations there is an absence of itching of the eyes, nose and roof of the mouth, which is frequently present in true hay fever. Also there would be a lack of an allergic history.

### HAY FEVER

Hay fever may be divided into three types according to the time of the onset (Northern and Eastern states)

1 Early spring usually occurring in April and early May, due to pollens from trees

2 Late spring or early summer usually occurring in May to August, due largely to pollens from the following grasses: timothy (*Phleum pratense*), sweet vernal (*Anthoxanthum odoratum*), June or Kentucky blue (*Poa pratensis*), orchard (*Dactylis glomerata*), redtop (*Agrostis alba* or *palustris*) and rye (*Secale cereale*)

3 Fall occurring about the middle of August until frost due to a large extent to the giant and dwarf ragweed pollens (*Ambrosia*)

The grasses causing the late spring or early summer hay fever named in their order of pollination are: sweet vernal beginning the first or second week in May, June grass the second or third week in May, orchard grass the third or fourth week in May, timothy, redtop and rye the first or second week in June. Pollination continues in all these grasses until about the middle of July.

Timothy is the most important of the grass pollens producing mid and late summer hay fever in the Northern and Eastern states. Bermuda grass and Johnson grass are the most important in the South Eastern states. In the Middle West broom grass and blue grass predominate.

The ragweed (*Ambrosia*) type of hay fever which makes its appearance in the early fall is the most severe and longest in duration of any of the seasonal attacks. On account of its buoyancy this type of pollen is very widely distributed. The pollen granules are carried even by light winds several miles from their source. In the Eastern part of the United States the ragweeds (giant and common) are the most abundant members of this family. The Western ragweed, sand bur and marsh elder are the most important causes of fall hay fever in the Western States with the wormwoods (mugwort, sagebrush) and *franseria* predominating on the Pacific Coast. In the Southwest the amaranths (careless weed and pigweed), salt bush, and the chenopods (lamb's quarters) are important during late summer and fall.

There are many localities to which these generalizations do not apply. In some districts other plants less common than those named above predominate.

Hay fever from grasses is more prevalent in England and on the Continent than in this country.

It has been estimated that the late spring and summer cases occur about eight times as frequently as the early spring type and the fall type about twice as frequently as the summer cases. About 5 per cent

APPROXIMATE POLLINATION PERIODS OF SOME OF THE HAY FEVER PLANTS IN THE NORTH  
ERN AND EASTERN UNITED STATES IN THE LATITUDE OF CHICAGO

	April	May	June	July	August	September
Elm						
Cottonwood						
Poplar						
Box elder						
Birch						
Oak						
Hickory						
Walnut						
Ash						
Sweet vernal grass						
Plantain						
Blue grass						
June grass						
Orchard grass						
Timothy						
Redtop						
Corn						
Pigweed						
Western ragweed						
Tall ragweed						
Short ragweed						

of the cases are attributed to the wind-borne tree pollens (particularly willow, hickory ash oak elm and black walnut). These cases are limited for the most part to a period of one or two weeks in the early spring. About 26 per cent of hay fever sufferers have the combined summer and fall types.

The various plants that are pollinated by insects such as clover dandelion, daisies honey suckle, roses and golden rod are not of great

importance as patients susceptible to these pollens are affected by intimate contact only. As a result attacks from these pollens are few in number and more or less mild in type.

**Symptoms** —The symptoms of hay fever are those of an acute coryza such as malaise, paroxysmal sneezing, serous discharge, headache etc. to which are added an itching in the region of the soft palate and the median palpebral commissures (inner canthi) of the eyes. The sneezing is accompanied by profuse lachrimation and serous nasal secretion and by congestion of the conjunctiva. The profuse serous discharge from the nasal mucosa may be followed by a contraction of the swollen mucous membrane which brings temporary relief.

The serous secretion from the nose is acrid, and excoriates the alae of the nose and the upper lip. The secretions may become sero-mucous and in some cases purulent in character if a secondary infection occurs.

Intermittent and alternate blocking of the nose are present. During the continuance of the nasal obstruction the patient suffers from the paroxysmal sneezing, headache and lachrimation.

The pharynx is often dry and painful upon deglutition. The tonsils are not usually inflamed although they may be.

Tinnitus aurium is frequently present and is due to a swelling of the mucous membrane of the eustachian tubes.

The appetite is impaired with a corresponding loss of weight.

Symptoms are worse on dry, sunny, dusty and windy days than on rainy days. They are usually worse in the early morning. They are aggravated by the proximity to the pollen and as a result are much worse in the country than in the city. There is relative freedom at the seashore excepting when there is a land breeze. There is usually complete freedom during an ocean voyage.

**Diagnosis** **Skin Tests** For routine tests the multiple puncture or scarification method is used but for testing the degree of skin sensitivity the intracutaneous procedure is essential. After washing the skin with alcohol and ether extracts of the various allergens are applied to different areas about 2 inches apart.

**The Multiple Puncture Method** —A sterile needle held nearly parallel with the skin is inserted in an oblique manner through the drop of pollen or other extract into the epidermis. Six to eight shallow stabs are made in order to carry the pollen extract into the deeper layers of the skin.

**The Scarification Method** —A sterile needle scarifier or a dull knife is used to abrade or scratch the outer layer of the skin about  $\frac{1}{4}$  inch long. The pollen extract is expelled from the capillary tube over the scratch and gently rubbed with the side of the tube.

If the cutaneous test is made with dried pollens a drop of normal salt solution or N/10 sodium hydrate is applied to each scratch. The dried pollen is then placed in the drop of liquid and gently rubbed in. One scratch is left with a drop of salt solution or sodium hydrate as a control.

**Intracutaneous Method** —In making the intracutaneous test which is the most accurate of all minute quantities of sterile fluid extracts of



the substance are injected by means of small sharp needles between the outer layers of the skin. Extracts from 1 to 10 000 000 to 1 to 100 are used depending upon the suspected sensitivity of the patient. If after trying 0.01 cc. of one of the more dilute extracts and a positive wheal is not obtained in five to thirty minutes a less dilute extract is

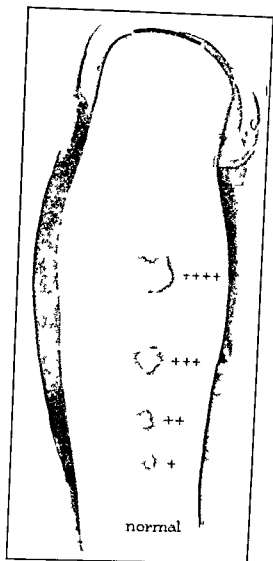


FIG. 84.—Allergic skin reactions varying from normal to 4+.

tried. A guide to the treatment is obtained from the degree to which the patient reacts to the various extracts.

**The Reaction.** If hypersensitiveness to the pollen protein exists within five minutes to one-half hour there will appear about the site

of inoculation a distinct urticarial wheel having a sharply defined but irregular border and often lasting for several hours. The wheel will vary from  $\frac{1}{2}$  inch to 1 inch in diameter with the elevated area more or less white and surrounded by a pink blush. The intensity of the reaction is usually but not always proportional to the anticipated severity of the clinical symptoms. Pseudoreactions are usually small regular and circumscribed.

**Ophthalmic Test**—In the ophthalmic test a small amount of the substance in a dry or extract form is dropped on the conjunctival surface of the lower eyelid. A positive reaction manifests itself within a few minutes by itching and burning of the eye. Hyperemia, reddening of the conjunctiva and at times nasal symptoms on the corresponding side.

**Intranasal Test**—In the intranasal test the allergen is dropped or sprayed into the nostrils. A positive reaction is shown by itching, sneezing, rhinorrhea and edematous swelling of the mucous membrane.

In contact dermatitis the patch tests may be used.

Reactions to tests with bacterial proteins may be delayed as much as twenty-four hours following their application.

Positive skin tests do not constitute a diagnosis. A careful history is of equal importance. The identity, distribution and habits of the hay fever plants of the patient's locality must be known. The pollen of insect-pollinated plants such as sunflower, golden rod, daisy, aster and all cultivated flowers is of little importance.

**Treatment—Pollen Vaccine**—The method of desensitization against pollen is by the administration of increasingly strong doses of an extract of the pollen or pollens to which the patient is specifically sensitive and in contact with so that the dosage ends after the time of the onset of the symptoms. It is preferable to continue throughout the season of heaviest atmospheric pollen concentration. Permanent desensitization is not produced as a rule so the process has to be repeated each season. There are all degrees of hypersensitivity to the various pollens so that every allergic patient requires careful individual study.

The pollen unit is the quantity of pollen toxin which can be extracted from the thousandth part of a milligram of pollen.

*The prenasal treatment should be started from six to ten weeks in advance of the attacks of hay fever so the desensitization may occur before the height of the pollination. The injection may be given at intervals of from a day to a week apart depending on the local and general reaction. A following treatment should be omitted until the reaction has disappeared from the preceding one. The amount of the antigen should be measured with accuracy using a special Dick or Schick syringe.*

The best guide to the intervals between and rate of increase of the injections is the response of the patient as evidenced by the local and constitutional reactions (or lack of reactions). In a very small percentage of the patients (probably 5 per cent) the beginning injection may be as low as 4 units and the maximum final injection may not be

higher than 1500 units; however, it is possible to carry most cases to 10,000 units or higher. The maximum dose of the prophylactic series should be reached at approximately the beginning of the hay fever season.

#### SUGGESTED DOSAGE FOR THE AVERAGE CASE

Dose	1-0.1	cc	of the	1 to 10 000	dilution,	10 units
"	2-0.2	"	"	1	" 10 000	" 20
"	3-0.4	"	"	1	" 10 000	" 40
"	4-0.7	"	"	1	" 10 000	" 70
"	5-0.1	"	"	1	" 1000	" 100
"	6-0.2	"	"	1	" 1000	" 200
"	7-0.4	"	"	1	" 1000	" 400
"	8-0.7	"	"	1	" 1000	" 700
"	9-0.1	"	"	1	" 100	" 1,000
"	10-0.15	"	"	1	" 100	" 1,500
"	11-0.2	"	"	1	" 100	" 2,000
"	12-0.25	"	"	1	" 100	" 2,500
"	13-0.3	"	"	1	" 100	" 3,000
"	14-0.4	"	"	1	" 100	" 4,000
"	15-0.5	"	"	1	" 100	" 5,000
"	16-0.6	"	"	1	" 100	" 6,000
"	17-0.7	"	"	1	" 100	" 7,000
"	18-0.8	"	"	1	" 100	" 8,000
"	19-0.9	"	"	1	" 100	" 9,000
"	20-1.0	"	"	1	" 100	" 10,000
Co-seasonal doses—	0.5	"	"	1	" 100	" 5,000

The co-seasonal doses of 5000 units may be repeated at weekly intervals until the height of the pollen season is past providing constitutional reactions do not occur.

Before the pollen solution is injected, the piston of the syringe should be withdrawn. If blood enters the syringe, it should be reinserted to avoid injection of the solution into a venule or capillary. An injection should not be increased or it may be advisable to reduce the amount if the previous treatment gave rise to even a slight constitutional reaction.

The *intensive* form of treatment may be given if the time is short. By the intensive treatment is meant the entire series of preseasonal injections given in a short period of time, that is, in the two to four weeks preceding the onset of the hay fever. This necessitates giving the injections daily or twice daily in the early doses and as frequently as the reactions of the patient will permit in the later doses.

The *perennial* treatment is especially indicated in certain sections of the country where there is more or less pollen in the air throughout the year. The treatment is continued after the pollen season at intervals of three to four weeks, with a below maximum dosage, depending on the patient's response. Before the pollen season starts the dosage is again brought to its maximum.

The *oral* administration of pollen seems to give limited immunity through the action of the injected pollen on the gastro-intestinal mucosa. Its ultimate status has not been determined as yet.

Treatment is preferably preseasonal but may be co-seasonal. The doses, expressed in Noon units, begin with 500 units and gradually increase until 240,000 units or more are taken. Much larger doses are

given orally than hypodermically. They are given twice a week as a rule with the main meal in which there is a liberal allowance of fat. From fifteen to twenty gradually increasing doses are usually necessary to reach the maintenance doses of from 60 000 to 240 000 units or more. The maintenance doses are given weekly.

Undue reactions (usually gastro-intestinal) are handled in the same manner as given for the hypodermic injections.

**Anaphylaxis** — A constitutional or anaphylactic reaction may occur in a small per cent of injections. It takes the form of a sudden general urticaria or an acute attack of asthma or hay fever.

It usually occurs within the first thirty or forty five minutes. Upon the appearance of the symptoms a tourniquet may be placed above the site of the inoculation and  $\frac{1}{2}$  to 1 cc. of 1 to 1000 epinephrine injected subcutaneously and repeated if necessary. Symptoms are relieved promptly as a rule. The tourniquet may be loosened and tightened at intervals over a period of one hour or until the symptoms do not recur.

**Palliative Treatment** Various local and internal remedies have been used for temporary relief.

Epinephrine is often successfully used. It should be applied to the nasal mucous membrane with a spray or with thin pledgets of cotton pasted over the surface of the mucous membrane. In an occasional case it excites irritation and sneezing.

Ephedrine occupies an important place in the treatment of hay fever. Its effect is similar to that of epinephrine but more prolonged. Ephedrine possesses the additional advantage of being effective when administered by mouth. Capsules containing ephedrine  $\frac{1}{2}$  grain and amylal  $\frac{1}{2}$  grain taken every two to four hours or until relieved have been effective for the relief of the symptoms.

Large doses of vitamin C (200 to 700 mg. daily) have had some favorable reports. Sodium bicarb. natc may be given with the ascorbic acid to reduce the acidity for sensitive patients.

Potassium chloride given 10 grains at a time five times a day has been reported upon favorably in recent literature in treating all forms of allergy including hay fever. Its exact value has not been determined as yet.

Alkaline solutions may be sprayed into the nose with temporary relief. In some cases a nasal douche with an alkaline solution is soothing.

The itching of the inner canthi of the eye may be relieved by the following prescription used as eye drops:

Ephedrine hydrochloride	m
Sodium borate	gr. v
Boric acid	gr. v
Camphor water	3 iv
Rose water	3 v

Calcium lactate powder or one or two tablets of calcium gluconate (20 grains) three times a day from one-third to one hour before meals is of distinct value in many cases. Ampules of sterile calcium gluconate solution may be used for intramuscular or intravenous administration.

Palliative relief of nasal obstruction may be obtained by the submucosal injection into the lower border of the inferior turbinate of a sclerosing solution such as 0.5 to 1 cc. of a 5 per cent solution of sodium morrhuate. The same result may be obtained by cauterizing the inferior turbinate with the electric crutery.

**Histamine Desensitization**—Histamine, a primary stimulator of the autonomic nervous system and capillary dilator, has been used in the form of histamine diphosphate to induce desensitization to histamine. Increasing doses of the substance is given for hay fever and other forms of allergy with good reports in many instances. The beginning subcutaneous dose is 0.1 cc. (or less) of a solution of histamine diphosphate equivalent to a 1 to 50,000 dilution of histamine base. According to Williams<sup>1</sup> this dose would not precipitate symptoms in the majority of cases. Subsequent injections are given twice daily, increasing each time by 0.1 cc. until symptoms or allergic signs disappear. The maintenance dose in most instances is 0.5 cc. of a 1 to 10,000 dilution of histamine base. Re-appearance of the allergic symptoms are common after discontinuance of the treatment.

Hapamine, a histamine-azoprotein without free histamine, has been found in a limited number of reports to be effective in treating allergic conditions minus some of the disagreeable systemic effects of histamine.

**Antihistamine Therapy**—Benadryl (beta dimethylaminoethyl benzhydrol ether hydrochloride), a new antihistamine preparation, decreases the vasodilating action of histamine. Reports from the Mayo Clinic (McLinn and Horton, Williams and Code<sup>2</sup>) in a limited number of patients suffering from hay fever and perennial vasomotor rhinitis indicate from 50 to 90 per cent temporary symptomatic relief. Symptoms tend to return within twelve hours after the drug is stopped. Benadryl seems to give symptomatic relief in the various other types of allergic reactions as well as hay fever. The average adult patient is given 50 mg. from two to five times daily. Some disagreeable side effects such as nervousness, vertigo or drowsiness have been reported in some instances.

Pyribenzamine hydrochloride has a therapeutic effect similar to benadryl. It is given to an adult patient in 50 mg. doses two to five times daily as necessary.

Niacin (nicotinic acid) has been used in the treatment of the various allergies. Williams<sup>3</sup> advocates a beginning subcutaneous dose of 25 mg. on each injection until a maximum of 100 mg. is reached. A maintenance dose of 100 mg. is given subcutaneously once a day for three months, then the same dose by mouth for another three months. In most instances it is necessary to continue the oral treatment to maintain relief.

**Ionization**—Ionization of the nasal mucosa for the relief of hay fever has been tried in recent years with disappointing results.

<sup>1</sup> Ann. Otol., Rhinol. and Laryngol. 53:397 (September) 1944.

<sup>2</sup> Proc. Mayo Clin. 20:417, 434, 439 (November 14) 1945.

## ALLERGIC RHINITIS    HYPERESTHETIC RHINITIS

**Synonyms.**—Perennial hay fever, vasomotor rhinitis, nasal hydrorrhea, and atopic coryza

There are a great many patients who complain of hay fever-like symptoms throughout the year, without seasonal periodicity. These individuals are irritated by substances other than pollen although pollen may be included in their sensitivity. As this subject has been covered under Allergy and Hay Fever a brief summary only will be given.

**Etiology.**—A common substance responsible in these cases has been orris root. This powder is an important component of rouges, face and hair cosmetics, as well as of various household powders. Other common irritants are house dust, feathers and insecticides. Certain foods such as wheat, milk, eggs, chocolate, tomatoes, string-beans, etc., are common offenders. A few drugs such as ipecac, aspirin, quinine, methyl salicylate, caroid, lycopodium, etc., may produce the symptoms.

Vasomotor cases have been divided into the allergic and non-allergic types. Many of the non-allergic types show some endocrine imbalance. The symptoms of the two types of cases are similar, and it is practically impossible to differentiate them clinically. The question of the relationship of internal secretions to allergy is therefore pertinent. It would seem that any undue taxation of the autonomic nervous mechanism through fatigue, toxemia, faulty metabolism, hypoadrenalism or hyperadrenalism will result in sympathetic reactions, of which vasomotor rhinitis and allergy are possible related expressions.

Allergic reactions are classified into four types, according to the mode of entrance of the exciting factor. The first type constitutes the inhalants, such as pollens, cosmetic powders (orris root), household dust, animal emanations, and grises of various natures. The second type of allergic reaction results from the ingestion of foodstuffs, as cereals, eggs, and milk. The third group is due to bacteria or their by-products, such as frequently accompany infections of the nose, tonsils or intestinal tract. This form is usually associated with abnormalities in blood chemistry, essentially a high uric acid and low calcium content. Duke has reported a fourth type, in which the patients are sensitive to physical agents, such as light, heat and cold.

**Symptoms.**—A history is usually obtained of frequent nasal infections with nasal blockage alternating from side to side associated with itching of the nose and palate at any time of the year. Many observers consider itching of the nose, throat, roof of the mouth or posterior pharynx as pathognomonic of hypersensitiveness. Frequently a family history of allergy, including migraine, urticaria, eczema, asthma or angioneurotic edema is obtained. Contact with any of the known allergens may precipitate an attack.

Rhinoscopic examination reveals a boggy mucosa and turbinate. The color is a pinkish-gray or a gray. As the hyperplasia and edema increase a polypoid degeneration may be observed. Microscopic examination of the nasal secretion usually shows an eosinophilia.

If the secretions are turbid and pus demonstrated a sinusitis or secondary infection may be considered to be present. Skin testing is of great help in establishing the diagnosis. The intradermal test is usually considered more accurate. Every case of non seasonal atopic coryza should be tested with the entire inhalant group including the pollens with the foods and with the bacteria.

**Treatment**—The immunologic treatment of cases of allergic rhinitis showing definite sensitization to the inhalants may be tried. If the patient is hypersensitive to any particular agent in his environment he should be protected if possible from contact with this particular substance. In many instances however it is impossible to eliminate the offending factor. In the latter event desensitization may be attempted by gradually increasing doses of the antigenic substance until tolerance is acquired. This tolerance which is obtained is only temporary in most cases and the procedure will have to be repeated at intervals of a few months to several years. In cases in which no sensitiveness can be demonstrated an underlying endocrine basis should be searched for and if such basis is established proper treatment instituted.

In the bacterial type cultures are made from the nasal and throat secretions and tested intradermally the ones that give positive reactions are used for treatment.

Histamine and the antihistamine compounds may be used in allergic rhinitis as listed under the treatment for hay fever.

Walsh<sup>1</sup> found the injection of alcohol into the sphenopalatine ganglion gave symptomatic relief in both the allergic and non allergic type of vasomotor rhinitis.

The administration of calcium salts with thyroid extract together with irradiation with the air-cooled ultra violet light as reported by Novak and Hollander may be efficacious in a few cases.

Non specific protein substances such as peptone, tuberculin, typhoid vaccine, milk, etc. may be tried as some favorable reports have been reported in the literature from this form of therapy.

Palliative relief is obtained from 1 to 3 per cent ephedrine sprayed into the nose as indicated.

## ASTHMA

**Etiology**—There are two types of asthma the allergic or extrinsic and the bacterial infective or intrinsic type. At times the two are combined. The first group is more common in adults and the second in young children and old persons. About 40 per cent of all asthmatic patients show allergic reactions. The percentage is greatest in children and young adults.

The exciting substances are of a great variety. The more common factors are the inhalants, pollens, animal emanations, household dust, cosmetic powders and foods. The most common foods are cereals, milk and eggs.

Bacterial infective asthma appears to be primarily of bacterial origin. It is not uncommon to see improvement in these cases if infected tonsils

or sinuses have been properly treated. The tendency to hypersensitive ness seems to be inherited in both types in a percentage of cases. The inherited tendency, however, is non specific as to the allergen involved. In very young children with extrinsic asthma the allergen is usually ingested. In adult life inhalation and contact are more important in initiating asthma. Frequently the asthma begins after an acute respiratory disease or a more or less protracted series of colds. A diagnosis of true bacterial asthma should be made only after elimination of all other possible allergic factors.

Hazletine believes that a toxic state is present in all cases of asthma and that this with an abnormally irritable sensory organ comprises the two producing factors in the spasm of asthmatic attack. Pollen asthma is usually aggravated by the inhalation of dust or irritating vapors by the odors of flowers, the essential oils or perfumes, by changes in temperature, changes in barometric pressure and by the quantity of moisture in the air.

Bronchial allergy due to sensitiveness to the various allergens may be brought out or made worse by acute bronchitis or rhinitis. The mechanism of the paroxysm is not clearly understood.

**Symptoms**—The characteristic symptom of bronchial asthma is an expiratory wheeze. Frequently there is the expectoration of thick tenacious mucus rich in eosinophiles. The wheeze is due either to the contraction of the lattice-like musculature surrounding the bronchioles to edema of the bronchiolar mucous membrane or to masses of sticky mucus in the lumen of the bronchioles. In less marked cases cough and shortness of breath on exertion may be the only marked bronchial symptom.

**Diagnosis**—The diagnosis of asthma is made by the history and the cutaneous reaction. The intradermal method of performing the cutaneous reaction is usually used as it is easier and more delicate. Associated allergic disturbances may be seen especially in children such as abdominal pain and cramping, anorexia, regurgitation, urticaria, edema, eczema and food toxemia.

Food allergy is indicated by a history of migraine, certain gastrointestinal manifestations of food allergy, asthma, urticaria or angio neurotic edema and a history of eczema in infancy. A carefully taken personal history and family history together with the skin test will usually enable one to make a diagnosis of the specific allergies, however all positive skin reactions do not mean necessarily the presence of allergies. The negative skin reaction in food allergy may be due to delayed clinical disturbances or the result of a cumulative effect over a period of days or weeks. If food allergy is suspected and the skin tests are negative an elimination diet may be used. In inhalant allergy the history is of great importance.

**Treatment**—The condition can be relieved in a large percentage of patients by avoidance of the substances to which they may be sensitive. When this is impossible or impractical specific protein treatment is justifiable. In specific protein treatment the offending allergen is given subcutaneously in infinitesimal doses to start with. The dose is increased



as rapidly as the patient's tolerance will permit and is continued until the patient gains enough tolerance to stand the amount with which his habits and environment bring him into contact.

The patient with pollen asthma in whom the cause cannot be removed may be treated the same as the patient with hay fever—that is with pollen extract injections.

In the bacterial type cultures taken from the infected focus and tested intradermally are used for the treatment. Good results are obtained from treatment with stock polyvalent vaccines autogenous vaccines or vaccine filtrates of the corresponding organisms preferably in a concentration of 5000 million organisms per cubic centimeter for the later doses. If one is unsuccessful in finding the allergen responsible for an illness symptoms can often be relieved temporarily by the use of non-specific protein therapy.

Histamine and the antihistamine compounds is given for the treatment of hay fever may be tried as for other forms of allergy.

As a temporary expedient the use of epinephrine (1 to 1000) or ephedrine (1 to 3 per cent) can be recommended. If epinephrine is used it is advisable to start with 0.5 cc. or less subcutaneously or intramuscularly given slowly. Doses of 0.2 to 0.5 cc. can be repeated at five or ten minute intervals until the patient is relieved. Massage of the site of the injection will prolong the action of the epinephrine.

Ephedrine may be used in the palliative treatment of bronchial asthma hay fever urticaria etc. The average dose by mouth is 0.025 gm ( $\frac{1}{4}$  grain) every four to eight hours according to the response. If this dose is not effective 0.05 gm ( $\frac{1}{2}$  grain) should be given. The dose is better given in water. Usually relief follows within thirty minutes after administration and persists for six to eight hours or longer. In children the doses should be in proportion to weight and age.

Surgical intervention on the sinuses of allergic children as a rule is not indicated except in exceptional cases. When the allergy is under control or in a quiescent period infected tonsils and adenoid should be removed.

### ACUTE CIRCUMSCRIBED EDEMA OF THE NOSE CORYZA EDEMATOSA

This affection may involve both the pharynx and larynx in the same case. It is not an inflammatory affection but is probably an edema of allergic origin. It is quite like urticaria though it involves the mucous membrane. It is usually associated with other symptoms or diseases as hay fever urticaria of the skin headache gastro-intestinal disturbances and itching.

The treatment should be directed to the allergic factor producing the circumscribed edema.

### NASAL HYDRORRHEA RHINAL HYDRORRHEA

Nasal hydrorrhea usually allergic is characterized by a thin watery and slightly opalescent secretion more or less serous in type. The amount

of discharge varies from a few drams to several ounces in twenty four hours. The secretion when tested with alcohol or acetic acid throws down a stringy precipitate like mucin. When the precipitate is boiled with dilute sulphuric acid a sugar like material is formed, this is probably due to the presence of mucin. The protein is coagulated by heat it does not reduce Fehling's solution. Peptones and proteoses are absent. The alcohol extract of the secretions contains no reducing substance. The secretion may be distinguished from normal cerebrospinal fluid by the presence of mucin and the absence of a reducing substance.

**Symptoms** — The clinical picture of nasal hydorrhea shades off in one direction into cases of allergic rhinitis with symptoms of intense local irritation while in the other direction it may consist of a passive and almost painless watery discharge from the nose. It is apparently a disease of adult life which affects males and females equally. Although it may be more marked on one side of the nose than on the other the flow usually comes from both nostrils. When handkerchiefs are soaked with it they generally become stiff when dry. In cerebrospinal rhinorrhea on the other hand the discharge is so watery that handkerchiefs dry quite soft and in this affection the discharge is limited entirely to one nostril unless there happens to be some obstruction on the affected side when it may make its way to the opposite nasal fossa. When the fluid is of arachnoid origin headache or other mental symptoms are frequent but are relieved by the discharge. The latter disease is not accompanied by lacerimation or suffusion of the conjunctiva and photophobia. It may occasionally give rise to sneezing especially in the morning.

In nasal hydorrhea the feeling of malaise begins with the discharge and only stops with its cessation. It is frequently ushered in with sneezing photophobia and lacerimation. It rarely continues during sleep while cerebrospinal rhinorrhea continues day and night. It is very erratic in its onset and in its intermissions and is very dependent on external influences and on conditions of health.

**Treatment** — The treatment should be addressed to the various etiologic factors found in hay fever or forms of hyperesthetic rhinitis or to any other pathologic condition present in the nose and accessory sinuses.

### CEREBROSPINAL RHINORRHEA

King in 1834<sup>1</sup> made a notable contribution to rhinologic literature when he described for the first time the escape of cerebrospinal fluid from the nose. Such cases had been previously regarded as nasal hydorrhea. It is characterized by a flow of cerebrospinal fluid by way of the nose.

**Etiology** — The possible etiologic factors are some pathologic change in the contents of the skull leading to increased intracranial pressure fracture of the anterior fossa of the skull involving the cribriform

<sup>1</sup> London Med. and Surg. Jour. 4: 823 1834.

plate, dehiscence in the walls of the sphenoid sinus pressure necrosis or ulceration from tumors of the brain or infection of the sinuses hydrocephalus internus a defect in the craniopharyngeal canal (forced open by an increase of cerebrospinal fluid) by way of the perineural sheath of the olfactory nerve and by way of the lymph passages from the arachnoid spaces

**Symptoms** —The fluid is clear and watery in contrast to the slightly opalescent and more viscid fluid of nasal hydrorrhea. The dripping is usually intermittent but may be constant. It is free from sediment odor albumin and mucin. It usually escapes from the same nostril day and night for many days. It may disappear and reappear suddenly. It reduces Fehling's solution and gives no precipitate (mucin) on adding acetic acid.

It is faintly alkaline in reaction and is either tasteless or slightly salty. The specific gravity is between 1.005 and 1.010.

**Pathology** —Various pathologic conditions have been found at autopsies which correspond in most instances to the various etiologic factors listed. In some cases no localized lesions have been found.

**Differential Diagnosis** —The differential diagnosis should be made from nasal hydrorrhea. The latter is bilateral as a rule and ceases during sleep. It also fails to reduce Fehling's solution.

**Treatment** —The treatment of cerebrospinal rhinorrhea depends upon the etiologic factor present.

The surgical approach to the cranial defect has been made both intradurally and extradurally. Transplants of the various fasciæ osteoperiosteum muscle etc. have been used to cover or plug the defect. Adson<sup>1</sup> obtains a wide exposure by means of a bifrontal osteoplastic craniotomy. German<sup>2</sup> utilizes a dural flap from the crista galli to cover a defect in the cribriform plate. If a fistula through the frontal sinus is present from a depressed fracture Dandy<sup>3</sup> elevates the depressed fragments of bone and sutures or covers the dural defect with fascia then replaces the fragments of bone. If a depressed fracture is not present he makes a unilateral frontal bone flap on the involved side. Whatever may be done extreme care should be exercised to avoid infection of the nose which might be communicated to the meninges or to the cerebrospinal fluid of the brain and spinal cord. Chemotherapy should be given as a postoperative prophylaxis.

<sup>1</sup> Ann Surg 114 697 (October) 1941

<sup>2</sup> Jour Neurosurg 1 60 (January) 1944

<sup>3</sup> Arch Surg 49 75 (August) 1944

## CHAPTER VIII

### HEADACHE NEURALGIA OF THE FACE AND HEAD NEUROSES OF ORIFACIATION

#### HEADACHE

**HEADACHE** a prominent symptom of many disorders is difficult to classify from either an etiologic or a pathologic standpoint. It may be associated with some generalized disease a prominent symptom of a disorder of an organ or a localized region or it may seemingly be independent of any disease. The cause or causes are frequently difficult to locate or evaluate and the mechanism of its production obscure in many instances.

**Etiology** Headache is an important symptom of almost all acute infections. A toxic type of headache may result from many chronic infections as well as from certain drugs or chemicals such as alcohol carbon monoxide or the sulfonamides.

Headaches due to disturbances of the vascular system are common and usually chronic. These headaches are most frequently encountered in hypertension migraine and histamine cephalgia.

Other causes of headaches are certain constitutional diseases such as gout and some blood diseases especially anemia leukemia and polycythemia. Headaches may be referred from or produced directly by such diverse conditions as allergic upsets endocrine disturbances intracranial diseases and growths orbital or eye pathology and nasal and sinus infections. Almost no region of the body is exempt from the possibility of producing a headache of some type.

**Types of Headache** The pain or headache may be extracranial such as the face or scalp or situated deep within the head either frontal parietal or occipital.

The extracranial discomfort is well localized as a rule and somewhat similar to surface pains elsewhere in the body. This type of pain or headache usually involves one or more branches of the fifth cranial nerve, the upper three cervical nerves or external auditory canal ramifications of the ninth and tenth cranial nerves. Any sensory nerve of the head or face may be involved in neuritis. Extracranial headache or pain may come from disturbances within or without the cranial vault.

If the lesion originates within the cranial vault and is above the cerebral tentorium the pain is usually transmitted by way of the ophthalmic branch of the fifth nerve. If beneath the cerebral tentorium, the pain or headache may be referred along the branches of the ninth and tenth cranial nerves and the second and third cervical nerves.

In areas anterior to a vertical line in front of the ears the sensation of pain is transmitted as a rule by way of one or more of the three branches of the fifth cranial nerve. Posterior to this line the painful stimuli are carried in most instances through the upper three cervical

nerves. However these generalizations are not of great help as an expanding brain lesion may have pain manifestations extending to any portion of the head.

The onset of a headache during the course of the day may help determine the type or cause in many instances.

Headaches beginning early in the morning or shortly thereafter are frequently associated with a frontal or maxillary sinusitis, brain dehydration from various causes, hyperinsulinism, large amounts of hypnotics, histamine cephalgia, cervical myositis or arthritis, hypertension with early renal failure and expanding intracranial lesions.

Headaches beginning in the afternoon or evening may be caused by the various toxins, nervous tensions, fatigue, eye disorders and in some instances by sphenoiditis and posterior ethmoiditis.

**Mechanism of Pain Transmission**—The brain substance, pia arachnoid, the ventricular walls, choroid plexus and the bone are insensitive to painful stimuli. The dura mater is sensitive to pressure and to pain when stimulated in the regions adjacent to the main arteries. The middle meningeal artery is quite sensitive as are the arteries forming the circle of Willis including about one-third of their distal length. Some of the venous sinuses and connecting cerebral veins are also pain sensitive (Ray and Wolf). The structures external to the cranial vault are sensitive to painful stimuli in varying degrees.

The mechanism by which head pain or headache is produced from any lesion within or without the cranial vault is by (1) direct pressure upon a sensory nerve usually in a late stage of the disorder, (2) a direct irritation or stimulation of pain sensitive nerve endings by bacterial agents, their products or other toxins, (3) excessive dilation or other abnormalities of pain sensitive arteries. These pain sensitive arteries are usually confined to the scalp and cranial cavity in the areas mentioned and rarely to other portions of the body, (4) and most important insofar as the cranial cavity is concerned is the traction or stretch placed upon the dura mater in the region of the large vessels of the pain sensitive portions of the vascular system by various intracranial lesions or abnormalities. This type of headache is independent of generalized changes in intracranial pressure (Kunkel, Ray and Wolf).

**Migraine**—Migraine is a periodic headache usually of twelve hours or longer duration with complete freedom from pain during the interim.

**Etiology**—A family history of migraine, allergy or epilepsy can be obtained in most instances.

The attacks usually begin in the second decade of life and are more common in women than in men. They are frequently worse during the menstrual period and may cease during pregnancy or at the menopause.

The theory that migraine is caused by an allergic reaction has received the most acceptance. That some disturbance of the endocrine glands, especially the gonads, is a factor in the etiology has also received much support. Abnormalities of the sella turcica with swelling of the pituitary

body and the hypoglycemic theory have been mentioned as etiologic agents. The theory that a neuropathy, psychopathic personality or emotional immaturity may be factors in the etiology has received some attention. The similarity of headaches of migraine and eye strain has been noted (Fridenberg<sup>1</sup>).

**Pathology.**—Migraine is a vascular disease on an allergic basis according to the general consensus of opinion. However, the exact mechanism by which the pain is produced is not so clear. There is strong evidence to believe that a primary vasoconstriction of the cerebral arteries occurs which produces the aura, scotomata and cortical sensory disturbances, followed by a vasodilatation which accounts for the pain by stretching the sensitive nerve fibers of these arteries (Schumacher and Wolf<sup>2</sup>).

**Symptoms**—The attack is preceded by an aura and usually by cortical sensory disturbances.

The pain is almost always hemicranial and frequently accompanied by pallor, dizziness, ringing in the ears, tingling or numbness of the face, tongue or arms, disturbances of taste and smell, frequency of urination or retention and diarrhea or constipation.

**Treatment**—Ergotamine tartrate, 0.25 to 0.50 mg. given as a hypodermic injection at the beginning of an attack, seems to be the most effective means of terminating the headache. It should be used cautiously in those patients presenting previous anginal symptoms due to the vasoconstricting action of the ergotamine. Dihydroergotamine (D.H.E.-45), similar to ergotamine tartrate, has been used (Horton, Peters and Blumenthal<sup>3</sup>) with good reports.

Potassium thiocyanate, a vasodilator, has been used, especially if hypertension is present. Oxygen inhalations (Alvarez), hypoglycemic reactions by insulin (Tillman) have been suggested. Endocrine therapy, based on the possible endocrine etiology of the headaches, has been used with some good reports. If an allergic history is present, the offending allergens should be identified and removed if possible or treated as for allergic reactions elsewhere in the body.

During an attack the patient should rest in a darkened room. Acetylsalicylic acid with or without codeine may help relieve the pain.

**Histamine Cephalgia**—Histamine cephalgia, also known as erythrocephalgia and neurovascular headache, is according to Horton<sup>4,5</sup> a clinical entity.

**Symptoms**—Histamine cephalgia is characterized by a severe unilateral type of headache, lasting less than an hour as a rule, beginning and often terminating suddenly. The headache tends to awaken the patient at night after one or two hours' sleep.

The constant, excruciating pain, boring or burning in character, involves the temple, eye, neck, frequently the face and at times may

<sup>1</sup> Med. Rec., 153:443, 1941.

<sup>2</sup> Arch. Neurol. and Psychiat., 45, 199, 1941.

<sup>3</sup> Proc. Mayo Clin., 20:241, 1945.

<sup>4</sup> Jour. Am. Med. Assn., 116:337, (February 1) 1941.

<sup>5</sup> Trans. Am. Acad. Oph. and Otolaryngol., (September-October) 1944.

extend to the shoulder or upper and lower teeth. In severe instances the attacks may occur every few hours day and night for months. The pain tends to follow the ramifications of the external carotid artery rather than the distribution of any cranial nerve.

Associated with the pain and on the same side congestion and profuse watering of the eye, stuffiness and watering of the nostril will be noted. Vasodilatation and increased perspiration and swelling of the temporal artery may be present. Nausea, vomiting and scotomata are absent. Heredity is not a factor in histamine cephalgia.

**Diagnosis**—The typical attack can be induced in most of these patients by the subcutaneous injection of 0.3 cc mg. of histamine base. The provocative attack should be differentiated from the immediate generalized headache frequently following a histamine base injection.

Histamine cephalgia should be differentiated from migraine, trigeminal and glossopharyngeal neuralgia and unilateral recurrent erosion of the cornea.

**Treatment**—The treatment consists in desensitizing the patient with increasing doses of histamine diphosphate<sup>1</sup> given subcutaneously twice daily. The first injection is 0.10 mg., second 0.15 mg., third 0.20 mg. and so on until about the ninth injection 0.50 mg. is given. At times up to 1 cc. can be administered. If flushing of the face or other symptoms of excessive response to the drug occur the next dose should be reduced 50 per cent and gradually increased from that point. To prevent future attacks a maintenance dose of approximately 0.50 cc. may be given two or three times weekly. In some instances a maintenance dose is not necessary.

The antihistamine compounds benadryl and pyribenzamine given in 50 mg. doses two or more times daily will give symptomatic relief.

**Temporal Arteritis**—In 1934 Horton, Magath and Brown<sup>2</sup> reported 2 cases of a syndrome which they called temporal arteritis. Cooke and his associates<sup>3</sup> reported 7 cases in 1946 and reviewed 31 cases that had been reported to that date. The disease seems to resemble the microscopic pathology of periarteritis nodosa in many respects. However, according to Horton and his associates the clinical picture is entirely different.

**Etiology**—The etiology is unknown. It is thought to be due to a focus of infection in most instances. Most of the reported cases have been in older individuals. Some of the attacks have followed the extraction of infected teeth. A clear conception of the pathologic process has been impossible to obtain.

**Symptoms**—The disease is characterized by temporal pain, malaise, fever, sweats and leukocytosis, and from two to six weeks later by a red, prominent or nodular temporal artery. Many of the reported cases had

0.275 mg. (1 cc.) of histamine diphosphate is the equivalent of 0.1 mg. of histamine base.

<sup>1</sup> Arch. Int. Med. 53:400 (March) 1934.

<sup>2</sup> Quart. J. Med. 15:47 (January) 1946.

an associated retinal arteritis with a decrease or loss of vision on the affected side

**Treatment** — A prolonged course with recovery is to be expected. Re-section of a segment of the temporal artery has been suggested in obstinate cases.

**Ocular Headaches** **Acute Glaucoma** — The headache from acute glaucoma is characterized by a sudden onset of a severe pain in the eye and head in the supraorbital region. The pain is worse at night and accompanied by blurred vision, dilated pupil, steamy cornea, shallow anterior chamber and increased ocular tension.

**Acute Iritis** — Acute iritis gives rise to pain radiating to the forehead and temple on the affected side. The pain is worse at night. The eye is active, red and with a small pupil. The iris is discolored and the tension normal. Vision is somewhat blurred.

**Acute Retrobulbar Neuritis** — The pain from retrobulbar neuritis is usually a unilateral discomfort or pain deep in the orbit, increased by rotation of the eye. The pain usually precedes the advent of blurred vision. A large central scotoma is usually present.

**Herpes Zoster Ophthalmicus** — A severe neuralgic pain on one side of the face and head usually precedes the typical herpetic eruption along the ophthalmic division of the fifth nerve.

**Errors of Refraction** — The headache from myopia, hyperopia, astigmatism and presbyopia may be a morning headache following an eye-strain from the preceding night or an afternoon or evening headache from strain during the day. Rest of the eyes relieves the headache.

**Heterophoria** — Heterophoria or imperfect muscle balance, if marked may produce a headache or pain in the eyes or other parts of the head. This may be accompanied by blurring of print, diplopia, nausea and vertigo.

**Headaches From Sinusitis** — Probably less than 5 per cent of all headaches are due to infections of the sinuses. Most headaches from sinusitis are due to an acute infection or an acute exacerbation of a chronic infection. Some headaches are due to a toxemia from a sinus infection the same as from a chronic infection elsewhere in the body.

The pain from an acute frontal sinus infection is greater in the morning shortly after arising. In the afternoon or evening the headache subsides or stops completely as a rule. The pain is aggravated by stooping or exertion. It usually increases each day for a few days and then as drainage is established and the acute inflammation subsides the intensity and duration of the discomfort decreases. In severe infections the headache may be constant but is usually worse during the day.

In acute maxillary infections the pain in the cheek may be of a neuralgic character involving the side of the face and upper teeth. Frequently the patient complains only of a full feeling in the cheek with a dull ache in the upper teeth. The pain is made worse by stooping or jarring.

In acute ethmoiditis a frequent complication of the ordinary head



cold the discomfort is usually expressed as a dull headache in the forehead or between the eyes. It is seldom the severe throbbing type found in acute frontal sinusitis. A similar headache may come from swelling and pressure in the superior and middle meati without involvement of the sinuses.

Headaches from acute sphenoiditis is usually a sensation of pressure or pain deep in the head or behind the eyeball. It is frequently referred to the occiput, neck, or to the supraorbital region and at times to the temporal area. The dull ache or pressure varies with the degree of inflammation and contained secretion within the sinus.

In chronic sinusitis the headache if present is indefinite and not well localized except when an acute exacerbation is present. The headache seems to be the result of a toxemia from the infected sinus rather than from the inflamed mucosa as in acute sinusitis. A neuralgic type of pain such as the sphenopalatine ganglion neuralgia may be transmitted along the ophthalmic branch of the fifth nerve in chronic sphenoid infections or possibly from chronic posterior ethmoiditis. A recurring migraine type of headache may also occur from a chronic hyperplastic involvement of the sphenoid and ethmoid sinuses.

**Endocrine Headaches**—Headache or head pain from an endocrine dysfunction is probable if a rise in intracranial pressure results usually because of increased water and salt retention.

A pituitary tumor or pituitary hyperplasia may make direct pressure on the surrounding structures producing a more or less constant pain in the frontal and temporal regions. The pain is usually bilateral and made worse by activity. At times the headache is occipital.

Tumors of the pineal gland may cause a severe brain tumor type of headache especially if the intracranial pressure is increased through blocking of the sylvian duct.

Headaches may be the result of disturbances of the thyroid gland. They are usually mild and with no characteristic location. Disorders of the adrenal gland may produce a headache probably from a salt and water retention. In hyperinsulinism headaches are probably the result of a hypoglycemia.

**Headache From Intracranial Lesions**—Headaches and the mechanism of their production from intracranial lesions have been considered in part in the first portion of this chapter and will not be repeated.

Headache may be an early symptom of intracranial pathology but may not be the principal one especially in intracranial tumors. When present in the latter condition the pain is usually sharp, severe and continuous or it may be a stabbing, boring, throbbing type. Changing the position of the head may bring on the pain or may give relief. Jolt, ing and straining tends to increase the discomfort. According to Pollock<sup>1</sup> the location of the pain is not characteristic and is not an index to the localization of the growth. Rapidly growing or hard or circumscribed tumors produce more pain as a rule than do their opposites. The head

ache from subarachnoid hemorrhage is a sudden, excruciating suboccipital type without hemiplegia as a rule. The severe pain of meningitis is accompanied by other signs and symptoms of the disease. Serous meningitis with paroxysmal hydrocephalus may produce a brief recurrent type of headache.

**Occipital Myalgia.** **Etiology** — A dull pain or headache in the occipital or neck regions is usually due to myositis or fibrositis of one or more of the neck muscles or their insertions. A cervical arthritis may be a factor in many instances. Nervous tension with hypertonicity of the neck muscles may also produce this dull pain.

Occipital myalgia usually follows an acute infection, chilling exposure to drafts, barometric changes or nervous overextension from any cause.

**Pathology** — Williams<sup>1</sup> in a study of 118 cases of myalgia of the head found the muscle involvement unilateral in nearly all instances. The muscles most commonly involved were (1) the upper border of the trapezius and its insertion, (2) the insertion of the splenius capitis into the mastoid process and that portion of the muscle just distal to the insertion, (3) the upper third of the sternocleidomastoid muscle, (4) the styloid process and the stylohyoid and anterior belly of the digastric muscle, (5) the styloglossus muscle and its insertion into the tongue with entire freedom from tenderness of the remainder of the tongue, (6) the superior constrictor of the pharynx (when the superior constrictor of the pharynx is involved, swallowing occasionally will produce a pain in the ear which is so severe that it is confused with glossopharyngeal neuralgia), (7) the crico-arytenoid posterior muscle (involvement of this muscle frequently produces pain on talking), (8) the temporalis muscle, and (9) the occipitofrontalis muscle.

**Symptoms** — Tenderness of the involved muscles can be elicited. The deep discomfort or ache may be referred along one or more branches of the fifth cranial nerve on the involved side. According to Williams, disease of the muscles does not result in increased subjective stiffness. The referred pain to the ear from myalgia of the superior constrictor of the pharynx is not relieved by cocaineizing the pharyngeal mucous membrane.

**Treatment** — Williams obtained relief in the majority of his patients by giving 100 mg. of mecin daily by hypodermic injections. The same dose given orally morning and night for two to five months maintained the relief.

The referred pain from occipital myalgia can be relieved by infiltrating a 1 per cent solution of procaine into the tender regions of the involved muscle.

Physical therapy in the form of heat, diathermy or deep massage to the neck muscles may be of value in many instances.

**Psychogenic Headache** — Psychogenic headache or a headache associated with a neurosis covers a number of sensations which may or may not be an ache or pain. Exaggerated terms such as terrible, intense, unbearable, etc. are frequently used in describing the

discomfort. The headache, usually continuous, may vary in location from day to day or hour to hour. According to Pollock<sup>1</sup> it is usually unaffected by weather or by ordinary changes in position. It is increased by work and the necessity to make decisions. It is relieved by reassurance or by the pursuit of some interest. Any new treatment or doctrine will abate the headache for a time. It is made worse by crowds, excitement or controversy. Other symptoms of a neurosis or a psychogenic disturbance will be noted.

Treatment is directed to the underlying cause of the psychoneurosis.

### NEURALGIA OF THE FACE AND HEAD

**Sphenopalatine Neuralgia.**—The sphenopalatine (Meckel's) ganglion (Fig. 4) is irregular in shape, about 1 cm. in length,  $\frac{1}{2}$  cm. in width, and  $\frac{1}{2}$  cm. broad; it is situated in the upper part of the pterygopalatine fossa. It is suspended from the maxillary nerve, second division of the fifth cranial nerve, by two or three branches and is in close relation to the posterior ethmoid cells, the lateral wall of the nose, and posteriorly in relation to the sphenoid sinus, if that sinus extends forward far enough. The fibers of the fifth nerve and those of the autonomic nervous system are in close relationship with the sphenopalatine ganglion. The Vidian nerve, formed from the great superficial petrosal nerve from the geniculate ganglion of the seventh nerve and the deep petrosal nerve from the carotid sympathetic plexus pass through the Vidian canal into the sphenopalatine ganglion. The branches of the internal maxillary artery are near the ganglion.

**Etiology.**—The exact cause or causes of sphenopalatine ganglion neuralgia has not been determined. It occurs in women about twice as frequently as in men, usually between the ages of twenty and fifty. It seems to have no seasonal incidence but is frequently seen when an acute nasopharyngitis is present. At times the condition is associated with the menopause. Sluder believed the irritation of the ganglion to be due to an infection or hyperplasia of the sphenoid or posterior ethmoid sinuses. Eagle<sup>2</sup> attributed the majority of his cases to an intumescence of the nasal mucous membrane with an associated intranasal deformity such as deviated septum, spur, ridge, adhesions, enlarged turbinate, growths, etc. Conditions of toxemia, anemia, fatigue, hysteria and emotional upsets are factors in the etiology at times. The exact mechanism by which the ganglion is irritated has not been determined.

**Symptoms.**—The clinical picture of sphenopalatine (nasal) neuralgia or "lower half headache," consists of pain about the eye, the upper jaw, and the teeth, extending to the zygoma and temple, with earache and pain in the mastoid, emphasized at a point about 5 cm. behind it. According to Sluder this point is always tender on pressure, although the pain is often temporarily absent. It may extend to the occiput, neck, shoulder, scapula, arm, forearm, hand, and fingers. There may be

<sup>1</sup> Ann. Otol., Rhinol. and Laryngol., 52, 730 (September) 1943.

<sup>2</sup> Ann. Otol., Rhinol. and Laryngol., 35, 66, (January) 1942.

also a sympathetic syndrome of sneezing rhinorrhea lacrimation and photophobia. The sense of taste is usually slightly diminished on the anterior half of the tongue and occasionally there may be vertigo.

Many neurologists doubt this syndrome and believe the explanation lies in the spilling over of pain impulses from the fifth nerve.

**Diagnosis** Cocainization of the sphenopalatine ganglion on the affected side with a 10 per cent solution of cocaine gives relief from pain within three or four minutes.

The differential diagnosis should be made from other unilateral neuralgic facial pains such as migraine trigeminal neuralgia temporomandibular joint syndrome lesions or disturbances of the teeth sinuses and orbit and from the various referred pains in general.

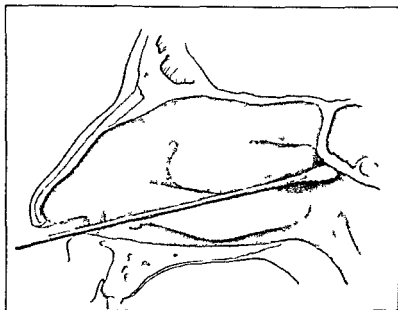


FIG. 85. Cocainizing the sphenopalatine ganglion. The cotton mounted applicator is moistened with a 10 per cent cocaine epinephrine solution and applied to the posterior wall of the nasopharynx immediately behind the posterior tip of the middle turbinate.

**Treatment**—These cases are extremely resistant to all forms of ordinary treatment but respond readily to cocainization of the sphenopalatine ganglion. Frequently one or more applications will give permanent relief. It is possible the relief of pain is obtained from the action of the cocaine on fibers of the fifth nerve.

If the disturbance is believed to be due to some intranasal pathology such as contact of a ridge or spur of the nasal septum with the middle turbinate or due to infection or hyperplasia of the posterior sinuses corrective measures should be taken. If some systemic disturbance or infection is a factor it should have proper attention. Permanent results are obtained in some cases with alcohol phenol injection of the ganglion.

*Injection of the Sphenopalatine Ganglion — Technique* — Anesthetize the posterior end of the middle turbinate and the wall just behind it with a 20 per cent solution of cocaine. Then Sluder's sword needle a straight needle  $5\frac{1}{2}$  inches long with a cross bar near the end is introduced from the septal side of the nose. The posterior end of the middle turbinate is transfixed and the needle pressed gently through the turbinate until the posterior wall is felt. The needle is then pushed upward outward and backward through the bony wall which is the anterior boundary of the pterygopalatine fossa in which the ganglion lies surrounded by connective tissue. Usually, by tactile sense one feels the needle slip into the cavity. The ganglion is about 0.6 cm. back of the wall. A 5 cc. Luer syringe filled with a 2 per cent solution of phenol (carbolic acid) in alcohol is then attached to the needle and from 5 to 15 minims are injected. If the needle succeeds in penetrating the ganglion the patient complains of excruciating pain in the eye ear top and back of the head and in the shoulder but should the phenol alcohol solution merely surround the ganglion the pain will be less severe.

The pain usually lasts anywhere from a few minutes to twenty four or forty-eight hours. The number of injections necessary is variable. If the ganglion is penetrated the first time as indicated by severe pain only one injection is required. If not two three or four attempts may be made at intervals varying from a few days to several weeks. If no relief is obtained after four such injections further attempts would be useless.

Resection of the ganglion has been tried in very severe cases but may not give complete relief in all instances.

**Vidian Neuralgia** — Vidian neuralgia is similar to sphenopalatine ganglion neuralgia. It is thought to be due to an irritation of the vidian nerve as it passes behind the posterior ethmoid cells and along the sphenoid sinus. It is usually associated with an infection of one or both of these sinuses.

Cocainization of the sphenopalatine ganglion does not give relief to the pain but relief is obtained from injecting 1 cc. of a weak solution of cocaine into the sphenoid sinus.

Treatment is directed to the infection of the sphenoid or posterior ethmoid sinuses.

**Nasociliary Neuralgia** — Greenfield Sluder describes a condition of pain in the eyes brow and root of the nose due to a neuralgia of the nasociliary nerve. The pain is usually referred to the small district bounded by the supraciliary ridge above the supraorbital notch laterally and the nasal bone below. Sometimes it extends to the tip of the nose. Inflammation of the anterior portion of the eyeball neuritic pains in the eyeball or orbital cavity and a unilateral rhinitis have been described as being associated with a nasociliary neuralgia. The nasociliary or nasal nerve descends into the nasal fossa through a small canal between the forepart of the cribriform plate and the frontal bone. After entering the nasal fossa it gives off internal or septal and external or lateral branches to the forepart of the nasal fossa.

This type of neuralgia may follow such infections as influenza malaria etc., or it may be secondary to an injury. At times it assumes a migrainous type of supraorbital pain which may alternate from side to side and occur in recurrent attacks. Long intervals of freedom may be noticed.

The diagnosis of this condition is made by applying 5 per cent cocaine solution on an applicator held up in the interior limit of the nasal fossa against the roof of the fossa. If the pain is of nasociliary origin this application of cocaine will stop it in a few minutes. Should it be of other origin it will not be influenced. If it is a supraorbital neuralgia the nerve in the supraorbital notch will be sensitive to touch. If of nasal ganglionic origin it will be relieved by cocaineization of the sphenopalatine ganglion. If the pain is due to sinus disease the diagnosis is made by the usual procedures followed in such cases.

Nasociliary neuralgia is usually a transitory phenomenon in the course of cases which have been under observation or treatment for something else. As a rule it is not obstinate.

**Trigeminal Neuralgia** — Trigeminal neuralgia (*tic douloureux*) is characterized by darting lincinating pains in the face. The intermittent attacks of pain involve the distribution of one or more branches of the trigeminal nerve. The second or third divisions separately or together are usually involved. Quite rarely the first division is involved alone. The attacks may increase in severity until the pain is more or less constant although remissions of months to years may occur.

The seat of the trouble lies in the Gasserian ganglion.

The etiology and pathology are undetermined. It is thought to be due to a discrete paroxysmal ischemia of the peripheral trigeminal structures. The average age of onset is about fifty years. It is believed that a septic neuritis of the dental or other nerve filaments may be a factor in the etiology.

Other conditions which may produce trigeminal neuralgia are tumors involving the fifth nerve or Gasserian ganglion, multiple sclerosis, syringobulbia, thrombosis of the posterior inferior cerebellar artery, chronicluetie basilar meningitis and chronic postherpetic neuralgia.

So-called trigger zones usually are present and these when touched may incite the sharp lancinating pains. Thus washing the face, brushing the teeth etc. may incite a paroxysm.

The differential diagnosis should be made from glossopharyngeal neuralgia, neuralgia or pain from a tooth, painful sinus infection and migraine.

The treatment consists of alcoholic injection of the trigeminal branches or division of the posterior sensory root of the Gasserian ganglion. The latter a major operation once regarded as a hazardous procedure with uncertain results has been perfected until now it is performed with safety and followed by cure. The alcoholic injection may not be permanent and subsequent injections may be required. The anesthesia produced by the first injections may last for about a year but subsequent administrations may have to be given with increasing frequency.

to control the pain. From 1 to 1.5 cc. of 70 to 90 per cent alcohol is usually sufficient at each injection. At times 3 cc. of alcohol are required.

The inhalation of 20 to 30 drops of trichlorethylene on a bit of cotton has been successful in some cases in lessening the pain.

Favorable results have been obtained in some instances by the administration of large doses of vitamin B<sub>1</sub> with or without the addition of concentrated liver extract rich in the anti-pernicious anemia principle.

**Glossopharyngeal Neuralgia**—This is almost identical with trigeminal neuralgia due to the fact that the ninth and fifth nerves supply contiguous sensory areas. The diagnosis is usually made after attempts to stop the trigeminal neuralgia have failed. The trigger zone in glossopharyngeal neuralgia is usually located in the tonsillar fossa with pains radiating to the ear rather than along the branches of the fifth nerve as in trigeminal neuralgia. The pain is instigated by swallowing, eating or irritation of the tonsillar region. Cocainization of the throat causes a cessation of the glossopharyngeal pain but has no effect on the pain from trigeminal neuralgia.

**Treatment**—The injection of alcohol for glossopharyngeal neuralgia is not recommended because of the close relationship of the ninth cranial nerve to the great vessels of the neck and to the tenth and eleventh cranial nerves.

Peripheral avulsion of the nerve will afford relief but the treatment of choice for severe cases is the intracranial section of the sensory root before it enters the jugular foramen. The nerve is exposed by a suboccipital craniotomy.

**Mandibular Joint Neuralgia**—Neuralgias and ear symptoms associated with disturbed function of the temporo-mandibular joint (Costens syndrome<sup>1</sup>) present an edentulous or partially edentulous mouth with changes of the joint from the wide overclosure of the jaw, so that undue pressure is made on the mandibular fossa with partial or complete closure of the external auditory canal.

**Symptoms**—The ear symptoms are an intermittent or a continuously stuffy sensation especially marked about meal time, tinnitus, dull pain within the ears and dizziness.

The pain may be a headache about the vertex and occiput and behind the ears. A burning or dry sensation in the throat, tongue and side of the nose may be noticed. The glossodynia and burning pains of the pharynx are from reflex irritation of the auriculotemporal and chorda tympani nerves to the lingual and glossopharyngeal nerves. The fifth nerve pain, however, is predominant.

Herpes of the external ear canal and buccal mucosa have occurred in a few cases. If the molar teeth are missing or the vertical dimension of the jaw is reduced the mandibular joint may show pressure changes.

**Treatment**—Inflation of the eustachian tube gives temporary relief to the stuffy feeling in the ears. If relief of the other symptoms is obtained by the patient wearing dental cork disks (2 mm. thick) or a dental splint

the diagnosis is more certain. Permanent relief is obtained by the correction of the overclosure with a suitable denture.

## NEUROSES OF OLFACTION

The neuroses of olfaction are characterized by either (a) a perverted sense of smell (parosmia), (b) oversensitiveness to olfactory stimuli (hyperosmia), (c) a partial loss of the sense of smell (hyposmia), or (d) a total loss of the sense of smell (anosmia).

**Parosmia**—Parosmia is characterized by a perception of imaginary odors, and may be due to pathologic changes in the olfactory brain-center. Inflammatory disease of the mucous membrane in the attic of the nose may also produce parosmia by overstimulating the nerve endings. It usually accompanies lesions of the central brain, although it occasionally occurs in hysteria, hypochondria, epilepsy, insanity, and syphilis.

**Hyperosmia**—Hyperosmia is characterized by an oversensitiveness to olfactory stimuli; that is, the perception of odors is exaggerated. The most delicate perfumes or odors not ordinarily perceived are recognized even to the point of unpleasantness. In some cases the perception of odors persists after the source of the odor is removed, and in this respect the condition approaches parosmia.

It may be due to an irritation of the olfactory lobes, hysteria, neurasthenia, hypochondria, sexual disorders in women (especially at the menstrual period) and to the lowered nervous forces accompanying wasting diseases.

**Hyposmia**—Hyposmia is characterized by a partial loss of smell, either from an impairment of the mucous membrane of the superior meatus of the nose, the nerve endings, the bulb, or the brain center. The impairment is only great enough to obtund the perception of odors without totally destroying it.

**Anosmia**—Anosmia is characterized by a total loss of the sense of smell, the pathologic lesion being more extensive than that found in hyposmia. A cold in the head, however, is a frequent cause of transient anosmia.

Odors reach the attic of the nose by either the anterior or the posterior nares, hence any condition of the septum or of the tissues of the outer wall of the nose which blocks the anterior or posterior nares may produce anosmia. The lesion may be in the nerve endings, as in atrophic rhinitis, in the nerve, or in the olfactory brain center. Anosmia of intranasal origin may be unilateral or bilateral according to the location of the obstructive lesion. In such cases the sense of smell may be restored by the proper medical or surgical procedures within the nose. If, however, the lesion is in the olfactory nerve or brain center, a cure is scarcely possible.



## CHAPTER IX

### CHRONIC INFECTIONS OF THE NASAL FOSSÆ

#### CHRONIC RHINITIS

**Definition**—Chronic rhinitis is characterized by a persistent unilateral or bilateral or alternating swelling or turgescence of the inferior turbinates. The patient complains of attacks of nasal obstruction and a thick mucous discharge.

It is questionable whether chronic rhinitis should be classed as a separate entity as the basic cause may be a low grade chronic sinusitis or an allergy; however many cases may have other etiologic factors in which disease of the sinuses may not be demonstrated or an allergic background discovered. It is those cases to which the following description may apply.

**Etiology**—The causes of chronic rhinitis are given under the etiology of acute inflammations and of acute rhinitis and will not be repeated in detail. It should be stated however that in many cases an undetected underlying allergy is the important etiologic factor. A secondary infection usually ensues which produces the thick mucous or mucopurulent discharge. A dysfunction of the endocrine glands especially a hypothyroidism may be a factor in many instances.

In a few instances obstructive septal lesions produce engorgement of the tissues without much irritation. The effect at first is one of turgescence which in the course of time results in hyperplastic rhinitis.

At times a chronically swollen inferior turbinate in one nostril may be explained by a complete or nearly complete blockage by a deviated septum on the opposite side. In order to maintain the nasal physiology the turbinate on the open side may undergo a compensatory swelling and overgrowth.

If in addition to the local turgescence there is an associated latent ethmoiditis the retention and decomposition of the secretions in the superior meatus and the ethmoid cells cause a prolonged low grade irritation which may result in a hyperplasia of the mucous membrane not only of the middle turbinate but of the ethmoid cells as well. Repeated attacks of acute rhinitis with a possible low grade or latent chronic ethmoiditis is a more common cause than is usually realized.

Dust, smoke-laden air, fumes and the overuse of nasal douching or spraying are common causes of this condition.

**Pathology**—In the early stage there is a distention of the venous or cavernous tissue of the turbinates with edema of the tissues. If the inflammatory process continues a true hyperplasia of the tissues takes place.

**Symptoms**—The symptoms are chiefly caused by transient stenosis of the breathway of the nose. In addition the secretions are heavier that is the mucoid element is increased while the serous element may

be decreased in quantity. The patient believes there is an actual increase whereas as a matter of fact, there is probably a decrease in the amount of secretion. The apparent increase is due to the greater consistency of the secretion which renders it less absorbable by the ingoing current of air. In a normal nose the secretions are comparatively thin or serous and are largely absorbed by the air current.

The transient stenosis is either intermittent or alternating, that is, both sides may be stenosed for a period and then open or the stenosis shifts from one side to the other.

**Treatment**—The treatment should be directed to the removal of the predisposing causes by the correction of a possible allergy, attention to the nasal or sinus infection if any exists and the removal or correction of any marked nasal abnormalities or other etiologic factors.

The transient stenosis may be controlled by ephedrine or if persistent by the use of the electric or chemical cauter.

### HYPERPLASTIC RHINITIS

**Etiology**—In the etiology of hyperplastic rhinitis allergens are important factors. Infection is usually considered a secondary development however some writers maintain the hyperplastic changes are due to a primary bacterial infection. The opinion that the inhalant and food allergens are the principal etiologic factors is based on the fact that eosinophilic cells are usually found in the membranes, nasal secretions and blood and a positive allergic history is obtained in the majority of patients.

The belief that the membranes are primarily infective is based on the fact that organisms are frequently demonstrated in the membranes and cases of true cutaneous sensitivity are seen that do not show hyperplastic changes.

Other factors may be long continued irritations from dust chemicals etc.

Nasal abnormalities such as marked septal deflections may be a predisposing cause in some instances. The sinuses especially the ethmoids may be diseased independently of the septal deviation and may thus be the primary cause of the hyperplasia. In either event the irritation, from dust smoke etc. or the irritation resulting from the secretions constantly flowing over the mucous membrane of the middle and inferior turbinates causes the pathologic changes in these structures.

**Pathology**—Hyperplastic rhinitis is characterized by thickened and edematous changes in the mucous membrane and periosteum. It is usually associated with polypoid masses of the soft tissues and rarefaction and osteoporosis of the bone.

**Symptoms**—The symptoms of hyperplastic rhinitis are often complex as the disease is frequently associated with an allergy or with a suppurative inflammation of one or more of the sinuses.

The symptoms arising from the hyperplasia are those of nasal obstruction, especially in the region of the inferior turbinate, that is, there is

more or less nasal obstruction and a sense of stuffiness or of pressure in this portion of the nose. The handkerchief is frequently used in efforts to dislodge the secretions and to overcome the sense of stuffiness. While the secretions may be thus removed the stuffy feeling often remains as it is due to overgrowth or congestion of the turbinates.

The secretions if present may be serous mucopurulent or purulent depending largely upon the complicating disease of the sinuses. However hyperplastic ethmoiditis from which polypi develop is not primarily a suppurative process. If a suppurative ethmoiditis occurs it is the result of a secondary infection as a rule.

Anterior rhinoscopy shows the middle or inferior turbinate to be enlarged paler than normal or it may be red and boggy and somewhat nodular in outline. Polypi may be seen growing from the middle tur-



FIG. 86.—The removal of the anterior end of the middle turbinate with nasal scissors.

binate ethmoid or maxillary ostium. Many of the ethmoid cells removed at operation may show a beginning polypoid degeneration. In view of these findings it is obvious that the removal of the visible polypi may fail to relieve the patient completely as the small budding polypi within the cells might later extend through the ostia into the nasal chambers. If the septum is deviated a ridge corresponding to the crista nasalis and the crest of the vomer may be present on one side while there is a bowing of the septum toward the opposite side in the region of the middle turbinate. The mucous membrane covering the septum is often thickened just below the inferior border of both the middle turbinates thereby obstructing both olfactory fissures.

If empyema of the posterior ethmoid cells is present pus may be seen in the olfactory fissure as well as in the lower portion of the nose. If there is hyperplastic ethmoiditis the anterior end of the middle turbinate may be red and boggy in texture. Patients with this type of ethmoidal inflammation at times complain of soreness or of fissures at the margins of the vestibules.

The obstruction in the upper part of the nose gives rise to a sense of

stiffness and of pressure across the bridge of the nose. These symptoms are rather constant as the tissue enlargement is permanent. There is also the additional symptoms of headache and vertigo—that is, headache in the frontal region limited to or more pronounced on one side and a feeling of soreness or tenderness of the eyeball upon ocular movements. The stooping posture increases the headache and temporary vertigo is often produced especially if the eustachian tubes are closed. The headache is also sometimes in the temporal, vertexial and occipital regions especially if the posterior ethmoid and sphenoid sinuses are involved.

In all instances of persistent sneezing attacks with profuse watery discharge from the nose we should suspect the presence of hyperplastic rhinitis or ethmoiditis and a careful inspection of the floor of the ethmoid should be made under the free edge of the middle turbinate.

**Treatment.** Complete sensitivity tests for allergy should be done and appropriate treatment instituted. Obstructive polyps, septal deviations or other obstructive nasal lesions should be given to any sinusitis or latent infection present. If nasal obstruction persists especially from hyperplasia of the inferior turbinate a submucosal or linear cauterization of the inferior turbinate should be tried. This will give relief in most instances. If much hyperplastic tissue remains along the inferior borders of the inferior or middle turbinates after adequate cauterization and obstructive symptoms persist the excessive redundant tissue may be removed by means of straight or curved nasal scissors or snare. Care should be taken to leave the turbinates themselves and the nasal mucous membrane in as nearly intact condition as possible otherwise marked disturbances of the nasal physiology might occur.

### Cauterization of the Inferior Turbinate

Various methods for cauterizing or scarring the intumescent or hyperplastic inferior turbinate have been used. One of the earliest methods was the linear cauterization by means of silver nitrate or chromic acid beads or trichloroacetic acid. These agents when successful frequently resulted in marked destruction of the nasal mucosa. Linear cauterization by means of the actual cautery is much more effective and does not have this objection to as great a degree. Diathermy or the high frequency current has been used for coagulation of the venous spaces with some good results but with severe reactions or sloughing in some instances. The submucosal injections of sclerosing agents has received much attention since Thacker's<sup>1</sup> report in 1940 in which a 5 per cent solution of sodium psyllate—a derivative of certain fatty acids from the seeds of the psyllium group seemed to be less irritating than sodium morrhuate and with less tendency to allergic reactions.

The submucosal injection of the fatty acid derivatives gives best results in chronic nasal obstruction from enlarged or engorged inferior

<sup>1</sup> Ann Otol Rhinol and Laryngol 49 939 (December) 1940

turbينات which still shrink well with astringents. If the hyperplastic process is well advanced with little response to vasoconstrictor drugs, the results are only partially successful as a rule.

**Submucosal Injection of Sclerosing Agents — *Technic* —** Anesthesia of the inferior turbinate is obtained by placing tampons or pledgets of cotton moistened with a 2 to 4 per cent solution of butyn sulfate above and below the turbinate. The tampons are left in place from five to ten minutes and then removed.

A 22 or 23 gauge steel needle about 2 inches long on a tuberculin syringe is used for the injection. A small strip of cotton is placed along the floor of the nasal cavity to absorb any escaping sclerosing fluid.

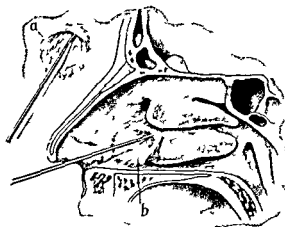


FIG. 87 — Method of applying the pledget of cotton to the inferior turbinate. *a* the pledget of cotton after being moistened with the cocaine or epinephrine solution is engaged upon the tip of a delicate probe. *b* the pledget of cotton being pasted or spread upon the inferior turbinate.

The needle with its beveled edge toward the septum is inserted into the anterior portion of the inferior turbinate at the mediosuperior angle and directed posteriorly along the bony portion of the turbinate to its posterior end. From 0.5 to 1 cc. of the solution is injected gradually as the needle is withdrawn. As a rule one turbinate only is injected at a time. The second turbinate may be treated a week later and the first turbinate may have the second injection, if needed, after two or three weeks from the first visit. Various reports indicate the procedure is a safe and satisfactory one in selected cases. It is not advisable to attempt injecting a sclerosing solution into the middle turbinate.

**Electrocauterization** — The technic of electrocauterization is as follows:

Induce cocaine anesthesia by the application of a 4 per cent solution of cocaine on a thin pledget of cotton to the swollen free border of the inferior turbinate for a period of ten minutes (Fig. 87).

Turn on the electric current until the point of the cautery electrode is of a bright cherry-red color.

Introduce the cold electrode into the nasal chamber and place it on the free border of the inferior turbinate (Fig 88). Then move it back-

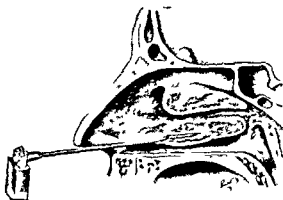


FIG 88 Lateral view showing the cautery electrode in position for cauterizing the inferior turbinate

ward and forward while still cold until sure of its correct position. Maintain the to-and-fro motion and press the contact spring of the cautery handle for one or two seconds when the contact should be broken. The to-and-fro motion should be continued until the electrode is cold that is for two or three seconds after the spring contact is broken and then it should be removed from the nose.

If these instructions are followed the procedure is painless and does not tear the eschar from the turbinate. If the to-and-fro motion is not maintained before, during and after the electrode is heated, the eschar will be torn off and the cauterizing effect lost.

The cauterization should be linear and should be about 1 inch in length. The whole length of the inferior turbinate may be cauterized in two or three sittings (Fig 89) never in one as too great a reaction and sloughing may follow.

The sittings should be from five to ten days apart. A week after the first cauterization the opposite side may be treated in like manner. At the end of another week the middle portion of the inferior turbinate

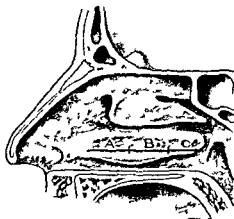


FIG 89—Showing the lines for linear cauterization on the inferior turbinate. A, B, and C representing respectively the first, second, and third cauterizations which should be made one week apart.

first cauterized may be thus treated. In many instances one cauterization over the most swollen portion may be sufficient.

The after treatment of a cauterized turbinate should consist in applications of a protective oil or ointment or an alkaline solution may be prescribed for home use. The alkaline solution should be used with a glass nasal douche rather than an atomizer as the force of the spray might injure the cauterized surface.

If the thick mucus persists the nose may be packed with small cotton pledgets saturated with a 10 per cent aqueous solution of ichthyol or a 10 per cent mild silver protein. Remove the pledget in about fifteen or thirty minutes and instil a protective oil.

**High Frequency Current**—The turgescient inferior turbinate may also be treated by coagulation of the venous spaces by means of the high frequency current.

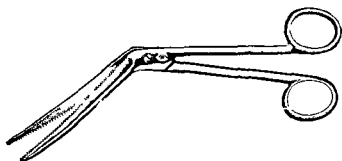


FIG. 90.—Serrated nasal scissors.

The monopolar method is used and coagulation is produced by inserting the needle for distances varying from  $\frac{1}{4}$  to  $\frac{1}{2}$  inch at various points in the submucosa and the current allowed to flow for about ten seconds. This is repeated in a week and again if necessary until the turbinate assumes a stationary position without obstruction.

**Surgical Treatment of the Turbinates**—The surgical removal of the middle turbinate is seldom indicated and almost never of the inferior turbinate.

**Hyperplasia of the Inferior Turbinate**—If the hyperplastic tissue of the inferior turbinate blocks the nasal passage and other methods of treatment have failed to reduce the hyperplasia the excess portion on the lower or free border may be removed by means of the serrated nasal scissors.

**Technic**—Induce local anesthesia by the application of a 5 per cent solution of cocaine by means of a thin pledget of cotton which should be placed over the hyperplastic area for ten minutes.

With nasal scissors cut off the necessary portion of the hyperplastic membrane.

If bleeding occurs the nose should be packed with tampons of vaseline gauze. If severe hemorrhage occurs from the posterior portion it may become necessary to introduce a postnasal tampon with Bellocq's

cannula (Fig 91) or by means of a rubber urethral catheter. A long strip of gauze should then be packed against it through the anterior nares. The tampon should be removed in twenty-four hours. It may be renewed if necessary.

**Submucous Turbinectomy** Under local anesthesia an elliptical incision is made over the most prominent portion of the inferior turbinate. This portion of the mucous membrane is resected. The excess mucous membrane and as much bone as necessary is removed. The flaps are sutured and a tampon saturated with bismuth and castor oil inserted if desired.



FIG. 91 Belloq's postnasal tampon cannula



FIG. 92 Removing the excess hyperplastic tissue from the lower border of the inferior turbinate by means of curved scissors. The turbinate bone is not removed.

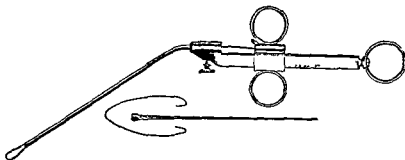


FIG. 93 —Krause's nasal snare

**Turbinectomy With Scissors and Snare** —The use of the scissors and snare is probably the most universally used method. It is safe and rapid when removing part or all of the middle turbinate.



*Technic*—Cocaine epinephrine anesthesia is sufficient

With Knight's or other nasal scissors sever the attachments of the anterior two-thirds of the middle turbinate as close to the outer nasal wall as possible

Engage the loop of a Kruse or other snare into the severed portion of the middle turbinate carrying the unengaged portion of the loop backward so as to include all or any desired portion of the middle turbinate. By tightening the snare the turbinate is severed from its attachment. It is removed by grasping with a suitable nasal forceps.

Packing may be used if bleeding is profuse

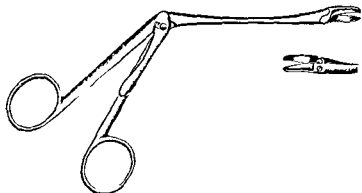


FIG. 94—Holmes middle turbinate scissors

With curved scissors of the Holmes or similar type the snare is not necessary as the blades are so curved that the cut made with them extends backward and downward until it emerges from the tissue (Figs 95 and 96)

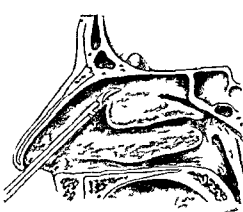


FIG. 95

FIG. 95—The removal of the anterior half of the middle turbinate with Holmes scissors



FIG. 96

FIG. 96—Anterior half of the middle turbinate removed with Holmes scissors exposing the bulla ethmoidalis

**Fracture of the Middle Turbinate**—In many instances removal of all or part of the middle turbinate is not necessary. Fracture of the middle turbinate away from the sinus ostia may give the necessary drainage and aeration.

**Technic**—After cocaine anesthesia the middle turbinate is grasped by a broad smooth bladed nasal forceps (Fig 97) and rotated in an inward and upward direction so that the inferior border and body of the turbinate is turned away from the outer nasal wall and toward the septum. A small strip of gauze may be placed between the inner portion of the fractured turbinate and the outer wall to keep the turbinate in the new position. The gauze should be renewed daily. Many times the turbinate will maintain its position without the use of the support.

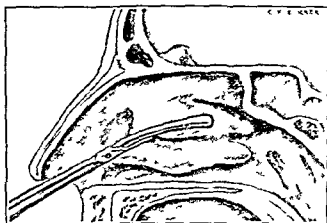


FIG 97—Forceps grasping middle turbinate preparatory to fracturing away from the outer nasal wall. The forceps are rotated in an inward and upward direction.

### CHRONIC ATROPHIC RHINITIS—OZENA

**Synonyms**—Chronic dry rhinitis, simple mucous rhinitis, mucopurulent rhinitis.

**Definition**—Atrophic rhinitis is characterized by a sclerotic change in the mucous membrane and occasionally of the underlying bone and by the presence of crusts and an offensive nasal breath.

**Etiology**—Many theories and hypotheses have been advanced in the explanation of chronic atrophic rhinitis. No one of the theories explains a sufficiently large number of the cases to be generally accepted. Any one of the theories will explain in a satisfactory manner some of the cases.

Probably one of the most extensively discussed theories is that of Grunwald's in which he claims the disease is the result of a suppurative sinusitis. The theory is that the mechanical and bio-chemical irritation from the sinus secretions causes a proliferation of the connective tissue cells which later contract thereby reducing the nourishment to the mucous membrane with atrophy and shrinkage as a later manifestation.

Possibly the next most discussed theory is that the disease is the result of some organism. This theory is very plausible and has many advo-

cates although the organisms held responsible vary from the spirochaeta pallida bacillus tuberculosis pseudo-diphtheria bacillus (Belfonti and Della Vedova) to the cocco-bacillus foetidus ozena (Perez) bacillus foetidus ozena (Hajek) Pes Gradenigo bacillus (Massei and others) bacillus mucosus (Abel) and other organisms

Other theories such as rarefying osteitis of the inferior turbinate (Cholewa and Cordes) or of the ethmoid (Lissier) or the developmental or structural changes such as the excessive patency of the nasal cavities in relationship to the shape or type of skull (Hopmann Siebenmann Gerber J Wright) or congenital narrowness of the nasal fossæ (Berliner Elliot Sauvage) or the arrested development of the inferior turbinate (Zaufal) may be mentioned but none of them have been proven Vogel associates the sphenopalatine ganglion with atrophic rhinitis in which he believed the ganglion shows changes of degenerative nature Fleishmann<sup>1</sup> believes true ozena is best explained by assuming a congenital inhibition of the development of the nasal mucosa which develops on the basis of a predisposition inherited according to Mendelian laws

The negro in Africa Oceania and the West Indies is free from the disease (St Clair Thomson) but the negro in South Central and North America may suffer from the affliction On the other hand the yellow races wherever found are very prone to have atrophic rhinitis

Inasmuch as the advent of the disease is usually at or before puberty is much more frequently found in women is more prevalent in some families than in others and is common in some races and absent in others it would seem as if some agent from within the body itself may be the determining factor in true chronic atrophic rhinitis rather than an external agent such as an organism trauma etc or the anatomic structure of the nasal cavities

Ozena occurs in from three to five females to every male The majority of cases occur about the age of puberty (ten to twenty years of age) however in many or possibly most of these patients a history of a nasal discharge for many years previously may be obtained A hereditary factor is present in many instances as the disease may be traced through several generations at times

The present trend of opinion is that the atrophy is due to an inherited factor and the crusting and fetor are secondary characteristics from drying through the wide nasal passages and from saprophytic organisms

**Pathology**—The bacterial flora of ozena is largely saprophytic The Wassermann reactions of the blood and spinal fluid are usually negative The disease rarely affects the larynx and trachea

The histologic changes of atrophic rhinitis are in the early stage a chronic inflammatory process and in the late stages a thickening and fibrosis of the arterial walls especially an obliterating endarteritis There is an early loss of the columnar epithelial cells and cilia The epithelium may undergo early stratification and a characteristic squamous-cell type of metaplasia In the late stage a considerable portion of the epithelium is of the squamous-cell type Subepithelially a dense fibrous tissue is present

The bone undergoes fibrosis and re-sorption the nerve structures undergo fibrous and degenerative changes and the glands are degenerated and replaced by undifferentiated fibrous tissue (fibricint<sup>1</sup>)

This atrophic-sclerotic process is never complete even in the advanced stages

**Symptoms**—The symptom complained of most bitterly is the odor or stench emanating from the patient's breath. The odor usually is not noticed by the patient as a partial or complete anosmia is present however it is most evident to others in the near vicinity, especially in the same room. The odor seems to be present in varying degrees depending to a great extent upon the amount of crusting present. If daily nasal cleanliness is practised the odor is lessened greatly or is absent.



FIG. 98. Chronic atrophic rhinitis (X 900)

Nasal obstruction due to the crust formation is complained of by nearly all patients. The crusts or dried secretions may fill the nasal chamber completely forming a cyst which may obstruct nasal respiration. If the crusts are removed a fluid mucopurulent secretion is found beneath and over the nasal mucosa. Frequently the crusts are so hard it is necessary to soften them before they can be removed. If attached to the mucosa as they frequently are in the anterior or other portions of the nares slight bleeding may occur. The crusting and odor may disappear after many years usually during or after middle life.

At times certain associated symptoms such as headache especially between the eyes or a stuffy or full feeling in the ears may be mentioned. Secondary or associated infections in the sinuses may occur or become active with the various symptoms of sinusitis resulting depending upon the location and extent of the sinuses involved.

**Examination.**—The examination of the nasal cavity reveals the greenish gray or grayish black dried crusts covering the turbinates and septum. The crusts may fill the nasal fossæ especially the inferior meati or may cover all or a portion of the mucosa without blocking the airway to any extent.

If the crusts are removed a marked atrophy of the inferior turbinate

becomes evident. A clear view of the posterior nasopharynx and the upper portions of the soft palate is obtained. The nasal mucosa has a pale shiny appearance.

**Diagnosis**—Chronic atrophic rhinitis should be differentiated from hereditary syphilis, foreign body with suppuration and crusting, chronic sinusitis and from marked anemia in which the erectile tissue of the nose is in a state of collapse.

**Treatment**—The treatment is essentially that of intranasal cleanliness. This is obtained by nasal douching at necessary intervals, usually from once or twice a day to two or three times a week. The douche may be administered by means of a douche bag, a fountain syringe, some form of a siphon douche or the Birmingham glass douche in which the flow is determined by a vent in the top of the glass container. The solutions used are usually a normal salt or sodium bicarbonate. The patient should be instructed in the manner of injecting fluids in the nose to prevent strangling with the danger of forcing the fluids or secretions into the sinuses or eustachian tubes.

To remove the crusts the nose can be picked with cotton saturated with a 10 per cent aqueous solution of ichthyol which should be removed in from twenty to thirty minutes. The crusts, being softened, are easily detached by blowing the nose or by the use of a cotton wound probe. This course of treatment if faithfully carried out will afford great relief.

Spraying the nose two or three times daily with an oily estrogenic solution for a period of several months has been tried by a number of investigators with some good reports. The exact value of this substance has not been determined as yet. The author (H. C. B.) has found an initial improvement during the first two or three weeks followed by a stationary period thereafter. It is possible the initial improvement is due to the increased attention such as the preliminary douching rather than to the estrogenic substance which follows the douching.

Mild astringent stimulating solutions may be of value in reducing the local infection. Any associated sinus disease should be treated as indicated. In recent years acetylcholine used locally with or without pilocarpine hypodermically has been advocated on the assumption that acetylcholine produces a vasodilatation and reactivity of the mucous glands.

Mixed vaccine made from various strains of the Perez bacillus has been advocated and used with little success. Tuberculin therapy (where acid fast bacilli could be demonstrated) has been used with some favorable reports.

Scarlet red has been used in recent years with variable results. Its action is based on its antiseptic quality and its stimulating action on cell proliferation.

Sugar treatment in the form of simple syrup has been used and many cases show improvement. The treatment consists in packing the nose with cotton or gauze strips saturated with the sugar solution and leaving in place fifteen to thirty minutes.

Implantations of ivory into the floor of the nose or septum or implantations of preserved septal cartilage on each side of the septum have been discontinued to a large extent.

### RHINITIS CASEOSA

**Synonyms** — *Coriza caseosa* *ozena caseosa* *rhinitis cholesteatomatosa* and *caseous purulent rhinorrhea*.

Rhinitis caseosa is a rare type of unilateral nasal disease characterized by the accumulation in the nose and sinuses of an extremely offensive cheese-like mass by the presence of a seropurulent discharge and other manifestations of chronic suppurative sinusitis and last by intranasal and frequently by extranasal deformity (Meversburg Bernstein and Mezz<sup>1</sup>).

**Etiology** — From their analysis of the material gathered in their search of the literature the authors mention the following theories on the pathogenesis such as the nasal erysipelas theory (Duplay) the scrofulous diathesis theory (Cozzolina) the disease entity theory (Streptothrix alba) the nasal cholesteatoma theory (Tilloux Wagner) the foreign body theory (Hill) and the necrobiosis of polyps theory (Bories and Schleicher). The usually accepted explanation is that the disease is secondary to a nasal stenosis which blocks the nasal discharge. Due to mechanical and chemical changes and continued exfoliation of the mucosa the bottled up secretion is transformed into the caseous material.

The condition is invariably unilateral. It is about equally divided between the sexes. It may occur at any age but the greatest number of patients are seen in the third and fourth decades of life. Most of the reported cases are French and Italian.

**Symptoms** — The early symptoms are those of a chronic nasal or sinus infection. Examination may reveal polypi or occasionally the presence of a foreign body. Cheese-like particles may be observed in the early stage. As the disease advances the discharge is more profuse and very offensive. Rhinoscopic examination reveals the characteristic cheesy mass with intranasal pressure deformity and erosions.

The late stage is characterized by facial disfigurement and discharging bony fistulas. The disfigurement consists of swelling of the cheek and widening of the bridge of the nose and at times a displacement of the eyeball forward and laterally. A fistulous tract in the canine fossa or frontal process of the superior maxilla may be present.

**Prognosis** — The prognosis is excellent with the complete removal of the cheesy debris and cleansing after-care.

<sup>1</sup> Arch Otolaryngol 23:449 (April) 1936

## CHAPTER X

### ETIOLOGY OF INFLAMMATORY DISEASES OF THE NOSE, THROAT AND SINUSES

**Inflammation.**—In 1872, Sanderson defined inflammation as “the succession of changes occurring in a part, as the result of injury, provided that that injury be not so excessive as to destroy the vitality of the part.” This would include inflammation due to infection, local and general immunity, phagocytosis and the repair of damaged tissue. Even the incidence of certain neoplasms could be included according to Cheate,<sup>1</sup> although he would exclude the repair and the formation of neoplasms from the subject of inflammation.

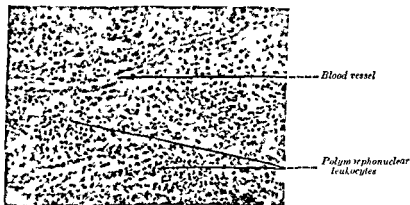


FIG 99 —Acute inflammation (× 200)

The general term, inflammation, may be defined as the local reaction of the body to irritation and consists in essence of the response of the polymorphonuclear leukocytes to this irritant. These mesodermal defense cells are gathered by a positive chemotactic response and pass through the blood-vessel walls by diapedesis to the site of the irritation where they may be seen microscopically (acute inflammation).

The cardinal signs of acute inflammation are “rubor, calor, dolor, and tumor.” In the area of irritation the blood-vessels (capillaries and small arterioles) following an initial contraction become dilated so that an increased vascularity of the part results, thus accounting for the heat and redness observed clinically. Next, in acute inflammation, exudation occurs, producing a swelling. The pain is due to the pressure of this exudate on the sensory nerve endings. If this irritant persists the reaction becomes chronic.

<sup>1</sup> Surg., Gynec. and Obst., 51: 310 (February 15) 1933

In chronic inflammation the essential reaction is not exudative but productive so that under the microscope much fibrous tissue can be seen. The older the lesion the greater the amount of fibrous tissue. In addition to the fibroblasts on microscopic examination lymphocytes, plasma cells and large mononuclears may be present.

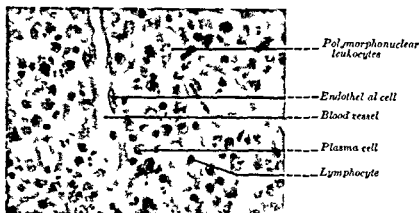


FIG. 100 — Cellular infiltration characteristic of both acute and chronic inflammation ( $\times 400$ )

In the so-called chronic granuloma the irritant stimulates the histiocytes to proliferate and these later may fuse to form giant cells.

Increased leukocytosis is an important reaction of inflammation. While the function and modes of activity of the leukocytes is not fully understood it has been fairly well demonstrated that the polymorpho-



FIG. 101 — Chronic inflammation with fibrosis ( $\times 700$ )

nuclear leukocytes, large lymphocytes and monocytes envelop and destroy bacteria while the macrophages which belong to the fixed cell type possess the ability of detaching themselves from the tissues and exhibiting their peculiar property of phagocytosis or enveloping and destroying broken-down cells.



The main distinguishing feature of the mucous surface is the presence of a layer of mucous cells of a glandular type capable of forming a large blanket of mucus. The importance of this moving blanket of mucus has not been realized until recent years.

Ilemming in 1921 found that the nasal mucus of a person with a severe cold yields a bacteriolytic agent given the name of lysozyme. It is peculiar to tears, nasal mucus and sputum but is more potent in the tears. Lysozyme probably plays a small part in the resistance to invasion of the upper respiratory tract.

**Etiology** — There are many predisposing causes of inflammatory diseases of the nose, throat and sinuses.

*Age* seems to exert some influence upon the resistance of the mucous membrane. Young children and young adults are more frequently subject to inflammatory diseases of the nose and throat than those of more advanced years. The lack of acquired immunity is probably the most important age factor. Other influences may be improper habits and insufficient protection of the body from the inclemencies of the weather. Persons of more mature years do not expose themselves needlessly as in youth and childhood.

*Sex* perhaps exerts some influence on the occurrence of inflammatory processes. Males are more frequently exposed than females hence they are more often affected by inflammatory diseases.

*Climate* undoubtedly influences the occurrence of inflammatory processes to a certain extent. In regions where there is much cold wet weather with sudden changes of barometric pressure and temperature it is more difficult to protect the body from the shock incident to such exposures. The shock thus sustained by the vasomotor nervous system leads to a lowered resistance of the mucous membranes especially of the nose, throat and accessory sinuses hence the growth of bacteria in these regions is favored. Recent investigations have shown however that climate is not as great a factor as once supposed. The incidence of colds in the native populations in various regions of the United States only varied about 10 per cent.

*Exposure* especially unusual or unequal exposure of the body to damp cold or other atmospheric conditions weakens the resistance of the tissues. The exposure of the feet to damp and cold is a most fruitful source of rhinitis and inflammations elsewhere in the body. Draughts striking a single portion of the body are detrimental to the resistance of the tissues much more than when the whole body is thus exposed. When partial exposure is experienced only a portion of the sympathetic and vasomotor mechanism is stimulated and an imbalance of the functional processes results the nasal expression of which is often some form of inflammation. Persons with focal infections in the sinuses or tonsils seem to be more susceptible to draughts and exposure than do those who are not carriers of infections.

*Lack of humidity and ventilation* in heated houses in the cold season are undoubtedly factors in predisposing to inflammatory diseases of the nose and throat.

*Clothing* is an important factor in maintaining or lowering the resistance of the mucous membranes of the upper respiratory tract. Too much is as productive of evil as too little clothing. If too much is worn the skin is rendered sensitive to slight exposure and if too little the body is subjected to continual stress and exhaustion of the vital forces results. Either condition prepares the soil for the growth of pathogenic bacteria in the respiratory passages.

Hard and fast rules cannot be laid down with reference to clothing as every individual is a law unto himself. The aim should be to so regulate the clothing as to avoid either extreme since to do otherwise subjects the system to shock and thus lowers the cellular resistance and prepares the soil for the growth of microorganisms and inflammation.

The *digestive tract* is regarded by many observers as contributory to inflammatory processes of the upper respiratory tract. If the processes of digestion and nutrition are imperfectly performed lowered resistance and a disturbed sympathetic system may result with reactions on the respiratory mucosa.

Certain *constitutional diseases* likewise produce a lowered resistance of the tissues including the mucous membranes. Diabetes, syphilis and all diseases due to faulty metabolism especially affect the tissues of the respiratory tract and predispose them to infection and inflammation.

*Heredity* probably has no direct influence in the predisposition to infectious and inflammatory diseases of the nose and throat. Indirectly it may have such an influence. That is, certain anatomic conditions or conformations of the tissues or structures of the nose or throat may be transmitted from parents to the child and thus establish a predisposition to infection and inflammation.

*Allergy* is a factor of importance in these inflammations as the allergic irritations predispose to many secondary infections.

Infected or enlarged *tonsils* or *adenoid* may interfere with the drainage and ventilation of the nose or inflammation focalized in them may lower the resistance of the mucous membranes of the nose, throat and sinuses and thus predispose to infection and inflammation. These and other extranasal influences may prepare the soil for the growth of pathogenic bacteria in the nose and accessory sinuses and result in inflammation of the sinuses without obstructive lesions in the nose. Whatever the cause of the lowered resistance of the mucous membrane the result is the same.

The *intranasal* predisposing causes of inflammation of the mucous membrane of the nose and accessory sinuses are perhaps best explained by the well recognized law. *Obstruction of the drainage and ventilation of mucous membrane lined cavities predispose them to infection and inflammation.* The character of the inflammation and the final result are partially determined by the location of the obstruction in reference to the various structures of the nose and the sinuses.

In summary it may be stated that the resistance of the mucosa to bacterial invasion depends upon a number of factors such as the moving mucous blanket, the action of the cilia, phagocytosis, the presence of

lysozyme in the mucosal secretions the local reaction of inflammation and the presence of a general and a local immunity. A decreased resistance to bacterial invasion may occur from improper diet from certain physiologic effects the result of chilling drafts improper ventilation mechanical or chemical irritations metabolic changes or from allergic disturbances. The types and properties of the various microorganisms would be factors of importance such as the effects of the toxic products the absence or presence of a capsule formation the adaptability of the organism to its environment in the various tissues or its ability to lodge in the intracellular spaces (Lenten<sup>1</sup>) and finally the obstructive effects of certain intranasal lesions which interfere with proper drainage and ventilation of the nose and sinuses.

<sup>1</sup> *Laryngoscope* 43 913 (April) 1933

## CHAPTER VI

### ACUTE INFLAMMATORY DISORDERS OF THE NOSE

#### ACUTE RHINITIS

**Synonyms** — Acute coryza common cold

**Definition.**—Acute rhinitis is an acute recurrent usually self limited inflammation of the mucous membrane of the nose and frequently the accessory sinuses characterized by chilly sensations lassitude nasal discharge and a swelling of the mucous membrane of the nose The patient also complains of a stuffiness of the nose and sneezing The upper respiratory tract is usually involved in addition to the nasal mucosa

**Etiology** — Acute infections of the upper respiratory tract are usually ushered in by the common cold Many different organisms have been suspected to be the causative agent The known pathogenic bacteria that are found in the nose and throat at most times are the various streptococci pneumococci staphylococci certain Gram negative cocci and *Hemophilus influenza* (Pfeiffer's bacillus)<sup>1</sup>

**Filtrable Virus** — The possibility that a filtrable virus is the cause of the common cold was first suggested by Kruse<sup>2</sup> in 1914 He reported the experimental production of colds by means of a bacteria free filtrate Foster<sup>3</sup> obtained similar results However many other observers have failed to confirm Kruse's findings Dochez and his associates<sup>4</sup> reported the experimental transmission of the common cold to anthropoid apes and human beings by means of a filtrable agent Their work on chimpanzees is suggestive experimental evidence that the etiologic agent of the common cold is able to activate pathogenic bacteria usually present in the upper respiratory tract such as the streptococci and pneumococci Further studies by this group<sup>5</sup> showed that the virus survives anaerobically in the cold for at least thirteen days it is inactivated at comparatively low temperatures by heat and lastly it has been demonstrated to multiply in tissue culture medium of the type previously employed in cultivation of vaccine virus

This apparent symbiosis of a filtrable virus with the known pathogenic bacteria of the nose and throat would explain the frequently seen clinical phenomenon of an initial mild rhinitis or pharyngitis later developing into a severe infection of the respiratory tract of the streptococcal or pneumococcal type

The various predisposing factors discussed in the chapter on The

<sup>1</sup> Deutsch med Wchnschr 18 98 1892

<sup>2</sup> München med Wchnschr 61 1547 1914

<sup>3</sup> Jour Am Med Assn 66 1180 (April 15) 1916

<sup>4</sup> Proc Soc Exper Biol and Med 26 567 (April) 1929 Jour Exper Med 52 701 (November) 1930

<sup>5</sup> Jour Am Med Assn 101 1441 (November 4) 1933

**Etiology of Inflammatory Diseases of the Nose Throat and Sinuses** would apply to acute rhinitis. A brief mention of the more common causes only will be given here. Acute rhinitis is more prevalent during the winter months. Children are more frequently involved than adults. Certain persons seem to be immune while others have repeated attacks. Immunity, if required, seems to be of very short duration as shown by repeated attacks in a short space of time.

Many writers and investigators have associated the common cold with an uncomplicated pandemic or epidemic influenza especially during the interpandemic periods. It is known that the same hemolytic streptococcus is frequently found in both conditions.

An enlarged adenoid or infected tonsils and adenoid predispose the individual to repeated attacks of acute rhinitis.

A chronic sinus infection is a factor of importance in the causation of frequent attacks of acute rhinitis. A preexisting chronic ethmoiditis which is more or less latent so far as symptoms are concerned is frequently found.

An occasional predisposing cause of acute rhinitis in adults is an obstructive lesion of the nasal septum which may impinge upon the middle or inferior turbinates thus interfering with drainage and ventilation of the nose and accessory sinuses. Septal deformity is not however a frequent cause.

All conditions local and general, which lower the resistance of the patient or the mucous membrane of the nose act as predisposing causes to infection and inflammation of the nasal mucous membrane.

**Pathology**—The vaso-constrictor muscle fibers of the capillaries are paralyzed and the dilator fibers irritated and as a consequence there is a hyperemia of the venous capillaries and lymph vessels and the nose becomes 'stuffed'. There is also an increased migration of leukocytes and a transudation of lymph and serum. The production of mucus is temporarily checked but later is increased. The epithelium is exfoliated and admixed with the other inflammatory products and secretions.

**Symptoms**—The symptoms are, for clinical purposes divided into three groups, as follows:

**First Stage or Onset**—The patient experiences a sense of dryness or prickling in the nose with itching at the inner canthi of the eyes. Chills, sensations and a feeling of malaise are usually present. Examination shows the mucosa to be red and hyperemic, but not fully turgescient. The mucous membrane is abnormally dry and free from secretions. Headache is usually present with a sense of fullness between the eyes. The temperature is normal or but slightly elevated.

**Second Stage**—This stage is characterized by a profuse serous discharge and turgescence of the mucous membrane. In some cases the headache and the sense of fullness between the eyes are diminished whereas in others they are increased depending upon the patency or closure of the ostia of the accessory sinuses and the impingement or pressure of the turbinates upon the septum. In those cases in which

there is a marked deviation of the nasal septum in the region of the middle turbinate the obstruction to drainage on one side may be more marked and the pain and sense of fullness correspondingly increased on that side

**Third Stage** — This stage is characterized by a mucopurulent or purulent discharge and by a decrease in the temperature if present. The headache and the sense of fullness between the eyes may be diminished to a dull heavy feeling across the forehead and between the eyes. If the nasal accessory sinuses are also involved in the inflammatory process the frontal headache and the sense of pressure are correspondingly pronounced. If the sinuses are not involved these symptoms may be entirely absent. Dizziness and vertigo may be present if the eustachian tubes are involved. A mild degree of acute conjunctivitis is usually present.

**Prognosis** — The natural duration of acute rhinitis is from three to ten days. When the sinuses are involved the duration may be extended to from two to several weeks unless the attack is aborted by appropriate treatment.

**Treatment** — The treatment of acute rhinitis would depend upon the action of certain drugs upon the ciliary action of the nasal mucosa, the nature of inflammation and the etiologic factors.

**Ciliary Activity** — Hilding<sup>1</sup> has shown that the ciliary action with a protective moving film of mucin over the surface are the chief mechanical factors involved in the drainage and defense of the nasal mucosa.

Lierle and Moore<sup>2</sup> found tap and distilled water slowed the ciliary beat. Three per cent ephedrine hydrochloride and 3 per cent cocaine hydrochloride were not detrimental to ciliary activity but at times increased it slightly, however 10 and 20 per cent solutions produced definite slowing with good recovery. Proetz<sup>3</sup> found 0.5 to 4 per cent of 2-aminoheptane sulfate without harmful action on the cilia. Mild silver protein of 5, 10 and 20 per cent produced an initial speeding followed by a slowing. A 1 to 1000 solution of epinephrine hydrochloride, 2 per cent zinc sulfate and 2 per cent mercurochrome were definitely detrimental and 5 to 10 per cent silver nitrate was fatal to the activity of the cilia.

**General Treatment** — The administration of 10 grains of acetyl salicylic acid at bedtime will induce rest and reduce the headache and muscular aches.

Diehl<sup>4</sup> reports good results from several doses of a combination of papaverine with codeine  $\frac{1}{4}$  grain each in the treatment of acute colds. This treatment does not seem to be of as much benefit in the subacute or chronic stages.

The administration of rhinitis or coriza tablets containing quinine belladonna and morphine, during the first stage will often stop the symptoms of acute rhinitis. However it is questionable if any beneficial

<sup>1</sup> Ann Otol Rhinol and Laryngol 41:52 (March) 1932

<sup>2</sup> Arch Otolaryngol 19:55 (January) 1934

<sup>3</sup> Ann Otol Rhinol and Laryngol 51:112 1942

<sup>4</sup> Jour Am Med Assn 101:2042 (December 23) 1933

effect on the rhinitis is produced other than the temporary suppression of the serous discharge. One tablet should be given every twenty minutes until dryness of the nose is produced.

Chilling should be prevented. In certain people with sensitive mucous membranes it may be necessary to reduce the amount of cold air admitted to the room at night for a few nights. This is especially true if free perspiration is induced as with acetyl salicylic acid. It has been demonstrated<sup>1</sup> that peripheral vasoconstriction with peripheral stasis and anoxemia lowered leukocyte response and impairment of the phagocytic action of the fixed tissue cells including that of the nasal mucosa are produced from chilling and draughts.

The value of alkalization in the treatment of upper respiratory tract infections has not been proved. However the free use of the citric acid fruits may be of value not only for their alkaline reaction but for their vitamin C content as well.

Laxatives should be given early in the attack if needed.

**Local Treatment—Epinephrine and Ephedrine**—The empirical use of drugs has long been practised and must doubtless continue to be practised until their action is better understood. We know enough about a few of them to criticize their use in acute coryza. Epinephrine has been much used in this disease because it was thought that the progress of the disease would be affected favorably by reducing the inflammatory reaction. Recent investigations would indicate that its use for this purpose is contraindicated except as a temporary measure to establish drainage and ventilation.

Ephedrine should be used instead of epinephrine as it has the advantage of more prolonged action with less after irritation and sneezing. It is used in a 1 to 3 per cent solution. The eiliary action does not seem to be interfered with in these strengths. The drainage and ventilation of the nasal cavities and the increased comfort of the patient warrant its use. The ephedrine may be used every two to three hours if necessary.

**Antiseptics**—In the first or second stage of an acute rhinitis good results may be obtained by first spraying a 1 or 2 per cent aqueous solution of ephedrine hydrochloride into the nasal chambers then after shrinkage has occurred swabbing spraying or snuffing a 1 to 1000 solution of merthiolate or a 1 to 3000 solution of metaphen over the nasal and pharyngeal mucosa. A second or third treatment should be given on successive days. This treatment is accompanied by a slight burning for a few minutes especially on the first day of the cold. Following the mercury antiseptic a bland oil may be introduced for additional comfort.

Excellent results may be had in the third or mucopurulent stage of acute rhinitis by placing a cotton tampon saturated with a 10 to 20 per cent freshly made solution of a mild silver protein in the middle meati just beneath the middle turbinates and leaving in place without blowing for fifteen to thirty minutes. The patient is more comfortable if a pledget of cotton is placed in the nares to prevent dripping. Two effects are

<sup>1</sup> Jour Am Med Assn 111 1744 (November 5) 1928

noted from this application, first the antiseptic action of the silver salt and second the "cathartic" effect on the glands of the mucous membrane which causes the glands to discharge their pus cells, bacteria and mucus.

**Infra red and Leukodescent Lamp**—In addition to the foregoing measures the use of the infra-red or the leukodescent lamp over the nose and face may be used. The infra red and heat rays exert a salutary effect upon the inflammatory process, that is, they increase the hyperemia and the leukocytosis. A treatment with the lamp should cover a period of from twenty to thirty minutes. It should be placed at a distance of about eighteen to twenty inches from the face. The eyes should be protected from the rays or an irritation of the conjunctiva may result.

**Vaccines**—The subcutaneous injection of heat killed bacterial vaccines may be of help in preventing a certain number of common colds.

The oral administration of an immunizing antigen (oral cold vaccine) seems to produce a heterophile antibody which may increase the resistance of many individuals to these upper respiratory infections.

Walsh<sup>1</sup> advocates the use of an intranasal vaccine spray as prophylaxis against the common cold as the best method at our command at present.

Vitamins A and D given together in large doses have been shown to have more value in preventing frequent colds than when either is given separately.

## STENOSIS AND CLOSURE OF THE LACRIMAL DUCT (DACRYOSTENOSIS) DACRYOCYSTECTOMY

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**Etiology**—The nasal conditions which have been considered responsible for dacryostenosis are (1) hypertrophic rhinitis (2) deflected septum, (3) tumors (nasal polyps) (4) ulcers (tuberculous and syphilitic) (5) injuries external or internal (operations upon the antrum) (6) sinus disease.

Only rarely, however, does examination of the nose in these cases show any of the pathologic changes here mentioned except perhaps a deviated septum which is very often deflected away from the diseased tear sac instead of towards it. The author (J. M. W.) has observed several cases following antrum operations (Caldwell Luc). Sometimes in the course of the intranasal tear sac operation it is anatomically necessary to open the ethmoid cells which are practically always found normal and on the other hand patients with sinus disease very rarely suffer from dacryostenosis. These facts indicate that the sinuses have little connection with stenosis of the lacrimal duct.

The etiology in most cases is perhaps best understood from the anatomy of the nasal duct. There is a dense plexus of veins similar to that of the turbinates situated between the mucous membrane and the bony

<sup>1</sup> Ann. Otol. Rhinol. and Laryngol. 49:8-5, 1940. Arch. Otolaryngol. 34:1093 (December) 1941.



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**Etiology**—The nasal conditions which have been considered responsible for dacryostenosis are (1) hypertrophic rhinitis (2) deflected septum (3) tumors (nasal polyps) (4) ulcers (tuberculous and syphilitic) (5) injuries external or internal (operations upon the antrum) (6) sinus disease.

Only rarely however does examination of the nose in these cases show any of the pathologic changes here mentioned except perhaps a deviated septum which is very often deflected away from the diseased tear sac instead of towards it. The author (J. M. W.) has observed several cases following antrum operations (Caldwell Luc). Sometimes in the course of the intranasal tear sac operation it is anatomically necessary to open the ethmoid cells which are practically always found normal and on the other hand patients with sinus disease very rarely suffer from dacryostenosis. These facts indicate that the sinuses have little connection with stenosis of the lacrimal duct.

The etiology in most cases is perhaps best understood from the anatomy of the nasal duct. There is a dense plexus of veins similar to that of the turbinates situated between the mucous membrane and the bony

<sup>1</sup> Ann. Otol., Rhinol. and Laryngol. 49: 875, 1940; Arch. Otolaryngol. 34: 1093 (December) 1941.

wall of the lacrimal canal. Swelling of these veins can readily close the duct. Repeated swelling of the veins eventually leads to a permanent stenosis and finally to complete closure of the duct.

**Symptoms** —The first symptom of dacryostenosis is tearing (1) epiphora. Later the tear sac becomes infected (usually pneumococcus) causing a (2) dacryocystitis (recognized by expressing pus into the conjunctiva on pressure over the sac) which may exist indefinitely unchanged or the sac may lose its elasticity and dilate developing a (3) mucocele (circumscribed painless swelling at the internal canthus) or a dacryocystitis may at any time break through the sac and form a (4) phlegmon (diffuse and very painful inflammatory swelling extending from the sac region), which through rupture or incision becomes a (5) fistula. Also in the presence of a dacryocystitis a corneal abrasion (foreign body) may develop into a (6) ulcer serpens often causing blindness. Thus dacryostenosis presents six different clinical pictures.

**Diagnosis** —**Test for the Patency of the Lacrimal Duct** —Having dilated (after cocaineization) the lower punctum with a conical sound introduce a lacrimal syringe and inject into the canaliculus boric acid which should flow easily in a stream from the nose (head inclined downward) if the lacrimal passage is freely patent. Should the nasal duct offer resistance to the injected fluid requiring force in closing the syringe and should the stream from the nose flow slowly or in drops (often of milky color) then the duct is obstructed. If no fluid at all escapes from the nose but returns through the upper canaliculus into the conjunctival sac the duct in this case is completely closed. Should it be impossible to inject fluid into the lower canaliculus the condition may be either a blocking of this canaliculus or a closure of both the nasal duct and the upper canaliculus. A fine sound introduced into the lower canaliculus will make the differential diagnosis. Folds of hypertrophic mucous membrane which approximate in the lumen of the duct and thus prevent normal drainage a physiologic closure may cause all the different clinical pictures of dacryostenosis although some fluid may still be forced with a syringe through the passage. Physiologic closure demands the same treatment as a complete stenosis.

When progressive inflammatory conditions of the tear sac advance sufficiently to change the contour of the region of the internal canthus the swelling (phlegmon or mucocele) or fistula is always situated below the horizontal axis of the eye. *This clinical fact is explained by the anatomic position of the ligamentum internum which crosses in front of and protects the upper part of the sac leaving the lower part covered only by the skin and subcutaneous tissue.* Hence increasing pressure within the sac will always cause it to bulge and finally perforate in the lower segment that is below the ligament which represents the horizontal axis.

**Differential Diagnosis** —The position of a fistula near the inner canthus is of diagnostic importance. Fistulas of the frontal and ethmoid sinuses perforate above the horizontal axis while those of the tear sac antrum and from a tooth (both very rare) appear below this axis. If

fluid injected into the canaliculus streams through the fistula, the diagnosis of a tear sac perforation is certain.

Occasionally other pathologic changes occurring between the sac and the skin are encountered, which distort the contour of the internal angle of the eye and in appearance have a marked resemblance to tear sac conditions, but which have no etiologic connection at all with stenosis of the nasal duct. Small cysts and tumors (fibrous) in the sac region may closely resemble mucocoeles in form, a spreading inflammatory swelling in the tissues surrounding the sac, a peridacryocystitis (Elschnig), may exist, and on inspection may be indistinguishable from a dacryocystitis phlegmonosa; a beginning epithelioma or a syphilitic lesion may simulate a lacrimal fistula. In all of these cases irrigation of the lacrimal passage demonstrates a freely patent duct and excludes a dacryostenosis.

**Treatment.**—The therapeutics of dacryostenosis in most eye clinics is as follows: Dacryocystitis is treated by irrigations of the sac and by probing the duct to restore its patency, and when the stenosis is not overcome (the usual result), the sac is then extirpated through an external skin incision, and in order to avoid the epiphora following the external operation, some ophthalmologists remove also the tear glands, which procedure in most cases does not cure the tearing.

The ophthalmologic treatment of fistula by curettage or attempts at extirpation of the sac are notoriously uncertain. One of the author's (J. M. W.) cases had undergone seven previous unsuccessful external operations by recognized authorities before being cured by the intra-nasal procedure to be described here.

Incision of a phlegmon evacuates the pus and relieves the acute symptoms, but still leaves the patient with his nasal duct obstruction and his dacryocystitis.

Of the methods of treatment here mentioned, probing, both painful and futile, is the only one which aims at removing the cause of the disease, namely the stenosis of the duct. The lacrimal canal, being a bony tube, cannot be dilated with probes. Probing only lacerates and mutilates the soft tissues within the bony canal and converts a physiologic closure (folds in the lumen) into a complete stenosis.<sup>1</sup>

Besides probing, there are surgical methods, external and internal, which aim at restoring permanent drainage between the eye and the nose. The external operation of Toti, "dacryocystorhinostomy," modified by Lagrange, Dupuy-Dutemps, Mosher and others consists in removing the inner half of the sac through a skin incision and then sewing the external half to the punctured nasal mucous membrane in such a way as to make a new communication between the sac and the nose. The clinical objection to all external operations is that, aside from the scar, the skin incision and the separation of the parts so alter

<sup>1</sup> There is one form of dacryostenosis however, that of the new born, in which probing is successful. In this condition the persistent fetal membrane closes the ostium lacrimale. When pressure on the sac fails to rupture the membrane, one single probing usually perforates the mucous membrane band, which then retracts leaving the duct patent.

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bone wall of the nose is laid bare over an area extending from the aperture pyriformis to the posterior border of the lacrimal fossa. The bone wall of the fossa is now removed with chisels (curved outwards) whereby the sac is freely exposed in the nose (Fig. 102). The sac is now grasped with a special toothed forceps pulled somewhat toward the nasal cavity and with a long scalpel the entire sac is excised. The mucous membrane flap is now returned to its original position and since it has somewhat contracted the outlet of the canaliculus is usually not covered. Should the flap extend over the mouth of the canaliculus a small section of the flap enough to free the canaliculus must be removed. A nasal tampon completes the operation. With the intranasal total extirpation of the lacrimal sac we have effected a direct communication between the conjunctival sac and the nose and have established clinically a conjunctivorhino-tomy. It is often very advantageous to do a partial submucous resection of the septum before beginning the sac operation.

**Indications**—The intranasal extirpation of the sac is indicated in all the various clinical conditions caused by dacryostenosis that is in dacryocystitis with or without dilatation of the sac in lacrimal fistula in phlegmonous conditions in epiphora of nasal duct origin<sup>1</sup> and also in *ulcus serpens* with dacryocystitis.

**Advantages of the Internal Operations**—1 The physiologic function of the lacrimal apparatus is reestablished so that not only a dacryocystitis a lacrimal fistula or a phlegmon is cured but subsequently the tears drain off into the nose and the troublesome epiphora usually following the external extirpation and often after the external dacryorhinostomy, is avoided.

2 The restoration of drainage from the eye causes the pathogenic bacteria to disappear from the conjunctiva which is very important when future intrabulbar operations (cataract etc.) are indicated.

3 The patient is spared the painful long continued and usually unsuccessful probing.

4 Removal of the lacrimal glands to cure the epiphora of dacryostenosis becomes superfluous.

5 An external incision or curettage which on healing tends to interfere with the drainage to the nose is avoided.

6 With the internal operation no external bandages are necessary.

**Totipot Moshier Operation**<sup>2</sup>—Moshier has modified Totipot's technique by using a straight incision 10 mm. from the inner canthus. The first opening is made through the lacrimal bone rather than through the superior maxilla. Totipot saves the mucous membrane before he makes the opening. Moshier resects it with the bone. Totipot does not remove the wall of the inner duct. Moshier removes it down to the upper rim of the inferior turbinate. Totipot occasionally removes the tip of the middle turbinate. Moshier does it in all cases.

<sup>1</sup> An epiphora may be concomitant with a dacryostenosis but have no causal relation to it. The intranasal operation would not be indicated in such a case. The differential diagnosis of this condition cannot be explained in a brief text book article.

<sup>2</sup> This portion of Dacryocystectomy has been prepared by the author (H. C. B.).

*Technic.*—A general or a local anesthesia may be used. If a local anesthesia is elected the novocain is injected along the line of the incision, above the inner canthus and down the inner wall of the orbit. In addition the nose may be packed with 20 per cent cocaine and epinephrine solution.

The lid, eyebrow and upper part of the nose are sterilized. The lids are held together with strips of sterile adhesive.

Any obstructing lesions of the septum should be resected and the anterior tip of the middle turbinate removed before the operation on the sac.

An incision (Moshé) is made about 10 mm from the inner canthus of the eye, starting at the level of the cartilage of the upper lid and extending down parallel with the posterior edge of the ascending process of the superior maxilla to within 2 or 3 mm. below the lower rim of the orbit. The knife is carried through the soft tissues and the periosteum to the bone.

The periosteum is elevated from the inner wall of the orbit by means of  $\frac{1}{4}$  inch flat chisel or a periosteal elevator. The periosteum is elevated to the rim of the orbit, passing onto the floor of the lacrimal fossa thus lifting the sac out of its bed.

The middle fossa of the nose is broken through and the opening enlarged by means of a small-sized biting forceps. The opening should be large enough to admit the little finger.

The nasal wall of the sac is removed by grasping the nasal wall with forceps or a small hemostat excising by means of scissors or a knife. The orbital wall of the sac is left in place.

The inner half of the wall of the sac is then removed with forceps and scissors. The outer half of the sac is left to protect the common opening of the canaliculi.

The inner wall of the nasal duct is removed by means of a small punch. The mucous membrane of the nose around the bone opening is trimmed, the soft tissues replaced and the skin sutured with dermal sutures.

Nasal packing and drains are unnecessary. The skin sutures are removed in forty-eight hours.

The after-treatment consists in instilling from 8 to 10 drops of petrolatum in the nose night and morning for from ten to fifteen days.

A contraindication to the Toti-Mc operation is the presence of a common duct of the canaliculi at its entrance into the lacrimal sac. This may be determined by passing a lacrimal probe into the sac or by roentgenogram.

## CHAPTER VII

### THE ETIOLOGY, PATHOLOGY, SYMPTOMS AND DIAGNOSIS OF SINUS INFECTIONS

#### THE ETIOLOGY OF SINUS INFECTIONS

The nasal accessory sinuses in man are the residual olfactory organs. In his primeval state the acute sense of smell was necessary, as it is in some lower animals. In the process of evolution the large distribution of the olfactory nerve has become less and less necessary, hence the sinuses are being gradually closed off from the nasal chambers until only small openings are present in man. Inflammation of the lining mucous membrane of the walled-off spaces becomes therefore a frequent pathologic process. If the sinuses were open more to ventilation and drainage inflammatory processes within them would occur less frequently, because the perpetuity and destructiveness of the process depend very largely upon the lack of normal ventilation and drainage. All the etiologic factors discussed under The Etiology of Acute Inflammatory Diseases of the Nose, Throat and Sinuses have an important bearing upon the etiology of sinusitis. A brief resume as it pertains to sinusitis will be given in this section.

**Etiology**—Hereditv is being recognized as having a much greater role than formerly thought possible. It is not a direct transmission of the infection but rather a tendency in which infection may occur under certain circumstances. The infantile sinuses may fail to clear of fluids promptly or the development of the sinuses may be arrested. Certain obstructive malformations of the septum, turbinates, uncinata, ostia, etc., may be inherited which may predispose to sinusitis.

In recent years an increasing stress is being placed upon diet and allergic states in relation to sinus infections. The importance of foods containing vitamins A and C has been demonstrated by clinical observations and laboratory research.

Among other general predisposing causes there may be endocrine or metabolic disturbances and lowered resistance from unbalanced or deficient diets, sepsis, toxins, infections, etc.

Among local predisposing causes are enlarged or infected tonsils and adenoid, hypertrophic nasal mucosa, pale boggy nasal mucosa associated with allergic conditions, enlarged or malformed turbinates, high septal deformities, cleft palate, atresia of the nares, traumatic or developmental deformities, trauma, foreign body and obstructing tumors. Impaired ciliary activity from any cause is an important factor.

Caries of the root of a tooth located beneath the floor of the maxillary sinus may cause empyema of the antrum by infection through the carious fistula thus formed, or by way of the vessels and lymphatics. It has been estimated that 10 to 20 per cent of all chronic empyemas of the



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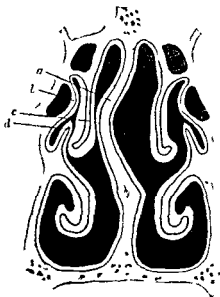


FIG. 103 — A high deviation of the septum causing closure of the infundibulum. *a* high deviation of the septum *b* inner wall of the bulla ethmoidalis *c* middle turbinate crowded against the outer wall of the nose and blocking the drainage of the infundibulum *d* uncinate process



FIG. 104 — Cross-section through the nose *a* hyperplasia of the middle turbinate which crowds upon the uncinate process *b* bulla ethmoidalis (*c*) and closes the infundibulum

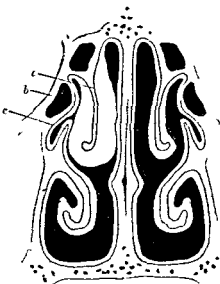


FIG. 105 — Edema of the mucous membrane of the middle turbinate blocking the infundibulum. *a* edematous middle turbinate *b* bulla ethmoidalis *c* uncinate process or inner wall of the infundibulum

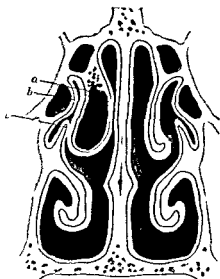


FIG. 106 — A large cell in the middle turbinate occluding the infundibulum. *a* cell in middle turbinate *b* the inner wall of the bulla ethmoidalis *c* the uncinate process or inner wall of the infundibulum or gutter

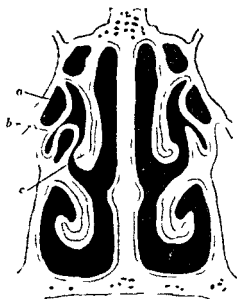


FIG 107 —Cell in the uncinate process (b) blocking the infundibulum a bulla ethmoidalis c middle turbinate

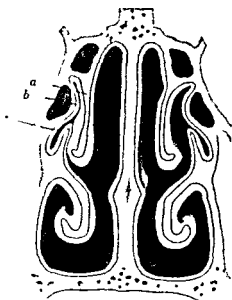


FIG 108 —The middle turbinate (a) clinging to the outer wall of the nose and blocking the infundibulum b inner wall of the bulla ethmoidalis c uncinate process or inner wall of the infundibulum

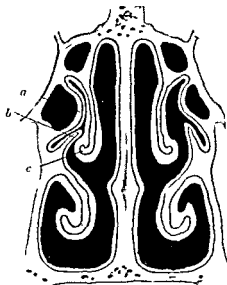


FIG 109 —Enlargement of the bulla ethmoidalis blocking the infundibulum a the inner and distended wall of the bulla ethmoidalis crowding inward and downward against the uncinate process and blocking the infundibulum b the uncinate process c the middle turbinate which on account of the bulging bulla appears to be the cause of the blockage whereas the bulla blocks

antrum have their origin in diseased teeth while the remainder are due to intranasal infections.

Nasal polyps are also regarded as a cause of sinusitis although the polyp itself is the result of a sinus infection especially the ethmoid and antrum or secondary to an allergic rhinitis. However this may be it is certain that the presence of a nasal polyp aggravates an existing sinusitis and its removal is often attended by an improvement of the sinusitis.



FIG. 110—Normal mucous membrane of the maxillary antrum. Magnified 63. Normal mucous membrane taken from the floor of the maxillary antrum shows a stratified columnar epithelium resting on a delicate basement membrane. The tunica propria consists of fibrous connective tissue which superficially is arranged somewhat loosely while deeper it is more compact and blends with the perosteum. The glands are found in the tunica propria and are most numerous near the ostium of the antrum (courtesy of Dr. Leroy A. Schall).

Foreign bodies in the nasal passages may cause sinusitis by erosion and subsequent infection of the nasal mucosa by directly blocking the cell openings or in rare instances by erosion through the adjacent nasal wall into the sinuses.

Nasal operations may result in sinusitis by reactionary infection and

inflammation which may extend directly through the outer nasal wall or via the cell openings into the sinuses. This is especially true if operations on the nose and throat are done while an acute rhinitis or tonsillitis is present.

Nasal dressings may cause a damming up of the secretions which undergo decomposition and infection and thus give rise to inflammation of the sinuses. Too much emphasis cannot be laid upon the untoward results of repeated intranasal packing as it is a fruitful source of inflammatory disease of the nasal and sinus mucous membranes.

Venous stasis from intranasal pressure may cause sinusitis. The pressure may be due to some anatomic or pathologic departure from the normal as tumors, foreign bodies, gummatous swelling of the septum, etc.



FIG. 111.—Chronic inflammation of the maxillary antrum. Magnified 2. The characteristic features are the immense amount of edema in the superficial portion of the stroma. The epithelium does not show much change. In places it is lacking which is due to an artifact in the preparation of the specimen. In the deeper portion of the tunica propria there is profuse round-cell infiltration with thickening of the blood vessels (courtesy of Dr. Leroy A. Schall).

It is probable that most persons succumbing to sinusitis after swimming are infected by their own nasopharyngeal germs mechanically driven into these cavities under conditions favoring rapid growth and retention of microorganisms.

Prevention of sinusitis from swimming would seem to be largely a question of warning those with acute rhinitis or other respiratory infections or with latent nasal infection—chronic colds and the like—to keep out of the water.

The exciting causes of inflammation of the sinuses are the various microorganisms causing the exanthematous and other infectious fevers. It is well known that the common cold and influenza are early phenomena in this class of cases. The inflammation usually extends to the sinuses where it may remain in a latent or chronic form. In some cases it is only after many years that the involvement of the sinuses becomes obvious enough to attract the attention of either the patient or the physician.



FIG. 112—Chronic inflammation of the maxillary antrum. Magnified 27. The epithelium is intact but with profuse round-cell infiltration of the stroma and proliferation of fibrous tissue. There is marked activity of the glands (courtesy of Dr. Leroy A. Schall).

It is probably true that the inflammation thus started is more likely to become chronic in those cases in which the cell openings are more or less blocked by obstructive lesions of the nose. If, for example, the septum in its upper portion is deviated to one side and lies against an enlarged middle turbinate, the sinusitis which develops during an attack

of one of the infectious fevers is more likely to continue into the chronic form than it is where no such obstructive deformity exists

### THE PATHOLOGY OF SINUS INFECTIONS

The pathologic changes which occur in the mucous membrane and bony walls of the sinuses in the course of suppurative inflammation are what might be expected in a mucous-lined cavity (Figs 110 to 115)

Infection of the nasal sinuses may occur in four different types—Acute congestive, acute purulent, chronic purulent and chronic hyperplastic

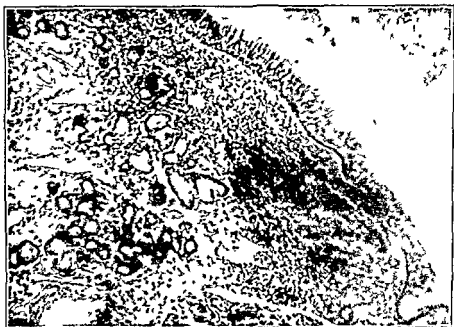


FIG. 113.—Chronic proliferative inflammation of the maxillary antrum. Magnified 63. Marked round-cell infiltration with superficial abscess formation. Marked activity of the glands and slight fibrosis (courtesy of Dr. Leroy A. Schall).

Chronic suppurative sinus disease may be classified microscopically into (a) edematous (b) granular or infiltrating (c) fibrous (d) a mixture of any or all of these forms. Connective tissue changes are common with much thickening in the subepithelial layer as a rule. This increase in cellular structure is composed of spiral, round, star-formed plasma, eosinophile and pigment cells.

For didactic purposes the changes which occur in the tissues may be studied in the following order which represents the usual sequence of the pathologic events.

(a) The submucous tissue is infiltrated with serum while the surface is dry. Leukocytes also fill the meshes of the submucous tissue.



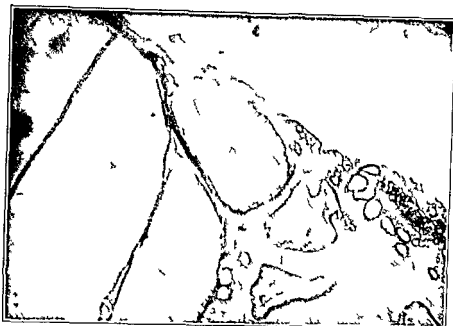


FIG 114—Cystic degeneration of the maxillary gland. Magnified 63 (courtesy of Dr Leroy A. Schall)



FIG 115—Chronic inflammation of the maxillary gland. Magnified 63. Much round-cell infiltration throughout the entire stroma with marked edema around the gland (courtesy of Dr Leroy A. Schall)

(b) The capillaries are dilated, and the mucous membrane is greatly thickened and red as the result of the edema and engorgement of the subepithelial structures. At this stage there is usually no defect in the epithelium.

(c) After a few hours, or a day or two, the serum and leukocytes escape through the epithelial covering of the mucosa, where they become admixed with bacteria, epithelial debris, and mucus. In some instances capillary hemorrhage occurs and blood becomes mixed with the secretions. The secretions, at first thin and watery, later become thicker and tenacious, on account of the coagulation of the fibrin of the serum.

(d) In many cases resolution by the absorption of the exudate and the cessation of the discharge of the leukocytes takes place in from ten to fourteen days.

(e) In other cases, however, the inflammation passes from the congestive to the purulent type, the leukocytes being thrown out in immense numbers. Resolution is still possible, although not probable, as the tissue changes are not yet of a fixed type. Unless the process is speedily arrested the tissue changes become permanent and chronicity is established. The underlying bone may show an osteitis and in places a definite bony necrosis.



FIG. 116.—Author's (W. L. B.) ethmoid curette

Extension of the infection from the sinuses to other parts may occur by: (1) a thrombophlebitis of the perforating veins, (2) by a direct extension through an ulcerating or necrotic portion of the sinus wall, (3) by way of a dehiscence and (4) through the vascular channels in the form of a bacteremia. It is questionable whether an infection may be transmitted from the sinuses by way of the lymphatics.

In chronic sinusitis the surface changes are similar to those in acute suppurative inflammation affecting other mucous membranes and bone tissue. The mucous membrane may present a granular surface, villous and fungoid excrescences, granular, cushion-like thickenings, etc. In the older cases there are hyperplastic thickenings. The membrane may be destroyed in spots by ulceration, exposing smooth, bare bone, or it may be soft or rough from caries. In some cases necrosis and bone sequestra are present, or they may be absorbed.

A microscopic examination of the sections of the mucous membrane sometimes shows a loss of the epithelium and glands, which are replaced by connective tissue. Ulcerations of the membrane are often surrounded by granulation tissue, especially if there is necrosis of the bone. Granulation buds may encroach upon the periosteum and thus unite the bone and mucous membrane. When this happens the bone is superficially absorbed and somewhat roughened in consequence. Osteophytes, or

bony scales or plaques resulting from plastic exudate sometimes form on the surface of the bone.

**Polypi in the Sinuses**—Polypi have been found in all the sinuses although they are more common in the antrum and ethmoid cells.



FIG. 117.—Showing the thin orbital-ethmoidal wall partially destroyed. During ethmoiditis this wall may be broken or perforated and give rise to orbital cellulitis.

They are much more common in the ethmoid cells than is generally supposed. Their hidden location within the small ethmoid spaces renders their diagnosis rather difficult. In the antrum however they are more easily diagnosed as they may be exposed through the canine fossa. A polypoid degeneration of the mucosa of the maxillary antrum may occur without any change in the normal appearance of the nasal mucosa even in the ethmoid region. The local symptoms depend upon the amount of hyperplastic changes which have occurred within the sinus cavity. As the hyperplasia spreads the symptoms such as serous discharge, occlusion of the nostril and sometimes sneezing become more pronounced, often simulating mild attacks of hay fever. When this hyperplasia becomes well advanced polypi begin to show themselves in the nasal cavities. As in hyperplastic ethmoiditis they may give rise to a recurrent polyposis of the middle meatus. This is in accord with Hirsch who believes this form of disease the most frequent cause

of recurrent nasal polypi. Iodized oil will be of great help in diagnosing this type of antrum pathology. Polypi have been found in the frontal and sphenoid sinuses although not so frequently as in the antrum and ethmoid cells. The polypi in the ethmoid cells are usually quite small on account of the limited space within the cells whereas in the antrum they are much larger.

The microscopic changes in the epithelium of chronic hyperplastic sinusitis are thickening, polypoid degeneration, metaplasia and areas of ulceration. The basement membrane shows thickening. The tunica propria reveals edema, round-cell infiltration, fibrosis and dilatation or compression of the glands and thickening of the blood vessel walls. The periosteum is thickened. The underlying bone may show osteoblastic or osteoclastic activity with fibrosis, hyperostosis, osteomalacia and necrosis (McMahon<sup>1</sup>).

In empyema of the ethmoid cells the thin lamina papyracea separating the cells from the orbital cavity may be perforated or entirely destroyed.

<sup>1</sup> Arch. Otolaryngol. 44, 1926.

by the suppurative process. The same is true of the cranial plate separating the cells from the anterior hemisphere of the brain. In the latter case the meninges are exposed to infection and may be the seat of meningitis, brain abscess or epidural abscess. Such an exposure of the meninges may exist in cases of latent ethmoid empyema with no other symptoms than a slight headache and mental irritability.

Fagleton has called attention to the tendency of the roof of the sphenoid sinus to perforate especially if the pneumococcus type III is the invading organism.

Thrombosis of the longitudinal and cavernous sinuses occasionally complicates sinus infection. Retrobulbar suppuration or orbital cellulitis is a comparatively infrequent complication.

In frontal empyema the floor and posterior wall are most often the seat of destructive changes.

### THE SYMPTOMS AND DIAGNOSIS OF SINUS INFECTIONS

The symptoms of sinus infections are divided into subjective and objective.

**Subjective Symptoms — Pain** — Pain referable to the region of the sinus involved may or may not be present. In active inflammation of the maxillary or frontal sinus pain is usually referred to the region involved. In the deeper sinuses as the posterior ethmoid and sphenoid the pain is vaguely deep seated in the head or it is referred to the periphery of the head without reference to the location of the sinus. For example sphenoid inflammation may give rise to pain in the occipital or to the frontal region. As a matter of fact inflammation in any or all of the sinuses usually causes pain in the frontal region. These pains are almost universally called headaches by the patient.

**Headache** — Headache is therefore one of the most common and significant signs of sinusitis though it may be present when the middle turbinate presses against the septum. This condition is often mistaken for eye-strain. Headache has multitudinous causes and is not therefore pathognomonic of inflammatory or other diseased conditions of the sinuses. Headache if due to eye-strain is usually bilateral and worse at the end of the day whereas in sinus disease it is more often unilateral or if not unilateral more pronounced on one side or it begins as a unilateral headache and extends to the other side. The headache which originates in a sinus is increased upon stooping forward and upon sudden jarring of the body. It may persist upon closing the eyes upon retiring or in a darkened room whereas if it is of ocular origin it disappears under such conditions.

The pains and headache due to disease of the frontal sinus may assume the form of sharp shooting pains through the eyes or they may be dull and heavy and nearly constant or they may consist of a dull feeling in the forehead which is aggravated by leaning forward. In the acute form of frontal sinusitis daily pain beginning at 10 or 11 o'clock in the morning and leaving by 3 or 4 o'clock in the afternoon is quite charac-

teristic. These attacks of periodic pain may last for a week or more. The patient generally complains of frontal headache which is limited to, or originates on, the side affected. Pressure under the floor of the sinus at the inner angle of the orbit usually elicits pain in these cases.

In the chronic form of sinusitis pain and headache may be absent unless drainage and ventilation are impaired.

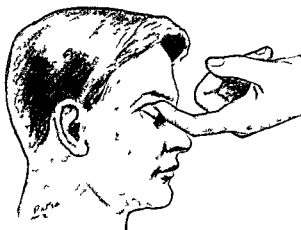


FIG. 118 — Pressure should be made under the floor of the frontal sinus. Pressure is often made under the supraorbital ridge whereas it should be made much deeper.

**Vacuum Frontal Headache** — A number of years ago Sluder called attention to the existence of what he called "the vacuum frontal headache." This type of headache is usually frontal, low-grade, and made worse by the use of the eyes for close work. The nose contains no pus and no pathology other than a possible redness or swelling of the nasal mucosa in the region of the middle turbinate. His explanation is, that obstruction in the inlet of the frontal sinus causes its contained air to be absorbed and a partial vacuum to be produced. More recently A. Hilding<sup>1</sup> has demonstrated that in a dog a  $-35$  more or less mm of water can be produced by ciliary activity. He excluded the possibility of blood absorption by obtaining the same results using an exsanguinated dog or a decapitated head.

Slight tenderness on pressure near the floor of the frontal sinus may be present.

The vacuum frontal headache is probably a clinical entity. There is as logical a reason for its existence in the frontal sinus as for a similar condition in the middle ear.

The treatment consists of shrinking the nasal mucosa and treating the nasal infection which is the usual causative factor.

**Tenderness Upon Pressure** — Tenderness and pain upon finger pressure may be present in disease of those sinuses contiguous to the surface of

<sup>1</sup> Ann. Otol. Rhinol. and Laryngol. 54: 725, 1925.

the face viz the frontal anterior ethmoid and the maxillary sinuses

For the examination of the frontal sinus pressure should be made over the anterior wall above the superior orbital ridge and under the floor of the sinus near the inner angle of the orbit

Tenderness over the frontal bone is rarely present in frontal sinusitis except in acute cases with obstructed drainage Tenderness is often present however when pressure is made against the floor of the affected sinus near the inner angle of the orbital cavity (Fig 118) The finger tip or thumb should be placed well under the roof of the orbit and the pressure directed upward Pain is thus often elicited even in chronic congestive cases Tenderness in this region does not however always indicate disease of the frontal sinus as the anterior ethmoid cells sometimes project beneath the floor of the sinus

When such an anatomic deviation is present the surgeon may be led to a wrong conclusion This difficulty may be obviated by having a roentgen ray film made as it will aid in determining the position and condition of the frontal and anterior ethmoid cells

In the examination of the anterior ethmoid cells pressure should be made at the inner angle of the orbit against the orbital plate of the ethmoid

In the examination of the maxillary sinus pressure should be made over the canine fossa of the superior maxilla

In sphenoiditis the anterior surface of the sphenoid sinus is sensitive to palpation and frequently as the examining probe causes pressure over the sphenoid sinus the patient will state that pain in the occiput or temporal region is excited

**Toxemia** — The various toxic symptoms such as malaise inability to concentrate muscular soreness stiffness of the neck etc may be present

**Disturbance of Equilibrium** — Giddiness and vertigo or a momentary sense of blurred or darkened vision and imminent fainting are frequently present in disease of the sinuses especially if the eustachian tubes are closed from an associated congestion A sense of fullness in the ears should accompany this condition All these symptoms may be aggravated or produced by stooping forward

**Disturbances of Smell** — The olfactory sense may be perverted (parosmia) the patient apparently perceiving odors that are not in evidence to normal noses A more common symptom is the loss of olfaction (anosmia) This is accounted for by the blocking of the olfactory fissure by the tissues in the region of the middle turbinate The ventilation of the superior meatus of the nose is thereby prevented hence the loss of the sense of smell In some chronic cases this may be due to the degeneration of the terminal filaments of the olfactory nerve although in most cases the sense of smell is regained after the infectious process has subsided

**The Objective Signs — Redness and Swelling** — When any of the sinuses contiguous to the skin of the face are involved (frontal anterior ethmoid or antrum) there may be redness and swelling covering the affected area If, for instance the frontal sinus is acutely inflamed there

may be swelling and redness in the frontal region likewise in the infra orbital region in antral disease and at the inner angle of the orbit in anterior ethmoid disease. When present this periostitis or edema gives the sensation on light palpation of a slight thickness or a velvety feeling over the affected sinus.

**The Nasal Wall** — The contour of the lateral nasal wall sometimes affords information as to the condition of the sinuses. In closed empyema of the antrum the inner wall of the antrum may be edematous or in rare cases pushed toward the septum. Likewise in empyema of the bulla ethmoidalis its median wall may be distended so as to close the hiatus semilunaris and impinge against the external surface of the middle turbinate.

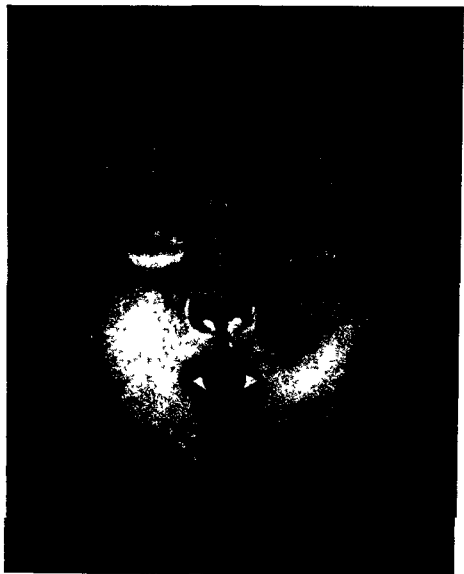
**Mucous Membrane** — The texture of the mucous membrane of the nose especially that portion of it covering the middle turbinate is sometimes indicative of sinus disease. That is when the mucosa of the anterior end of the middle turbinate is boggy and velvety in texture it usually signifies the existence of an acute inflammation of the ethmoid cells. If hyperplastic an associated hyperplasia of the mucous membrane of the ethmoid or maxillary sinuses may be suspected.

**Nasal Discharge** — Pus within the nasal chambers is usually significant of empyema of the sinuses. The nasal mucosa is rarely the focal center of suppurative inflammation whereas the sinuses are commonly the focal center of such an inflammation. The presence of pus in the nasal chambers should therefore excite suspicion of the existence of an inflammation of the sinuses. In a general way it may be stated that pus in the middle meatus signifies an involvement of the frontal anterior ethmoid or the maxillary sinus as these cells drain into the middle meatus. If pus is seen in the olfactory fissure (between the septum and middle turbinate) the posterior ethmoid or the sphenoid cells are presumed to be involved as these cells drain into the superior meatus above the middle turbinate. Pus is usually absent from the nose in hyperplastic sinusitis.

**Transillumination** — Transillumination of the sinuses affords objective information as to the condition of the maxillary and the frontal sinuses but none in reference to the other sinuses.

In transillumination of the maxillary sinuses the Freer-Jackson or other light is placed in the mouth in a dark room with the patient's eyes open. If a sinus is normal three points should be noted namely (1) The red pupillary reflex (2) the crescent of light corresponding to the position of the lower eyelid and (3) the sense of light in the eye when closed (Plate III). If the red pupillary reflex and the crescent of light are absent the antrum is probably affected. Note both sides at once and thus determine which one if either is affected. A comparison of the lower portion of the field of illumination may be very misleading as the anterior wall of the antrum varies greatly in density irrespective of the disease present. The orbital or upper wall of the antrum is however more nearly uniform in its density in all cases and affords a fair opportunity for a comparison of the transilluminated light through the two orbital plates that is when both orbital plates of the antrum are

# PLATE III



Transillumination of the Antrum

Right side normal : e pupillary reflex and crescent of light present Left side diseased.



normal the amount of light transmitted through them is about equal whereas when one is thickened by an inflammatory exudate the transmission of light is interfered with hence the crescent of light is dimmed or altogether absent. Likewise when both orbital plates are normal (antral disease absent) the light transmitted into the interior of the eyeball is shown in the red pupillary reflex in each eye whereas if one antrum is involved the pupillary reflex is absent upon that side and present on the other. The sense of light (eyes closed) is present on the healthy side and absent upon the diseased side in maxillary diseases.

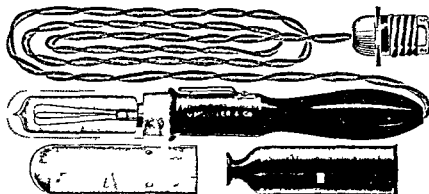


FIG. 119 Greer-Jackson transilluminator

The antrums may be transilluminated from above by placing a small light above the supraorbital ridge and slightly within the orbit. The beam of light is directed downward through the orbito-antral plate. The reflected light is viewed over the palate by looking through the mouth if the sinus is healthy. If a thickened mucosa or secretions are present in the sinus the transmitted light is absent or diminished.

Transillumination of the frontal sinuses is a satisfactory means of diagnosis in adults with well-developed sinuses. In children with small sinuses or in adults with bilateral involvement transillumination is not so reliable. The hooded lamp should be placed under the floor of the frontal sinus at the upper and inner angle of the orbit and the two sides compared. If the lamp is not placed well under the supraorbital ridge the skin transmits the light and may thus lead to a false deduction.

**Roentgenography**—Roentgenography of the nasal accessory sinuses is essentially a shadow transposition of the differential densities of the bones of the skull as modified by certain diseases or conditions such as

1 *Age*—In infancy there are numerous modifying conditions—for example size shape development etc. of the sinuses and bones of the skull. In infants and children roentgenography as well as transillumination are very unsatisfactory due to the absence of or slight development of the sinuses.

2 *Sex*—Men are apt to have thicker or heavier bones than women.

3 *Disease*—Such as loss of lime salts.

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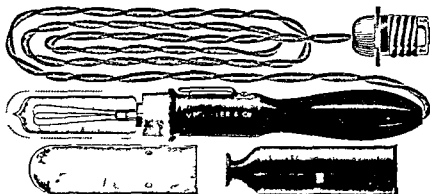


FIG. 119. Freer-Jackson transilluminator.

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2 *Sex*—Men are apt to have thicker or heavier bones than women.

3 *Disease*—Such as loss of lime salts.

4 *Thickness of Bones of Face and Skull*—This may be a localized thickening a unilateral or bilateral thickening

5 *Asymmetry of the Sinuses or Bones of the Skull*

6 *Altered Mucous Membrane*—This may take the form of an excessive thickening due either to infection past or present as a postoperative result or the alteration of the mucous membrane may be an extreme thinning or atrophy

7 *Inflammation or Suppuration within the Sinuses*

8 *Inflammation or Suppuration Adjacent to the Sinuses*

9 *Tumor Formation Involving the Region of the Sinuses or the Sinus Itself*

10 *Angle of Exposure*—Various dense structures such as the petrous portion of the temporal the vertebrae or the base of the skull may be projected in line with the sinuses obscuring and interfering with the proper reading of the films

In view of the above modifying conditions it is illogical to assume that a diagnosis can be made from the films alone. A secondary place perhaps should be given to the roentgen ray and the main reliance should be placed on clinical means. Caldwell was the first to show that the contents of a sinus whether pus normal salt solution or water offer about the same degree of obstruction to the roentgen ray. Beebe arrived at the same conclusion after injecting the sinuses with liquids of varying densities from water to thick pus. Differentiating between a pus-filled sinus and a neoplasm is not always easy although the neoplasm usually involves other structures as well which somewhat simplifies the diagnosis. Radiopaque oils give additional information in differentiating.

The angle or plane of exposure is important and should be governed by the sinus or sinuses suspected. The custom largely prevalent in the past is to have one sagittal (posterior anterior) view and one lateral view. When the expense is to be considered this perhaps will give as much general information as any other two views but the objection to these is that in the lateral view the ethmoids and sphenoids are superimposed upon the opposite sides and their outlines are confused or blurred. In the posterior anterior views the ethmoids and sphenoids are superimposed and an intelligible reading frequently rendered impossible.

An excellent procedure when general information concerning all the sinuses is desired is to have three views one posterior-anterior (Caldwell) and two obliques (Rhese).

In the posterior-anterior view the tip of the nose and the forehead touch the film. The rays are passed from near the occiput at right angles to the film.

The right and left oblique views are taken as first described by Rhese. In the oblique position the object is to project the sphenoids and the posterior ethmoids through the orbit. To accomplish this the tip of the nose, the brim of the orbit and the malar bone touch the film and the rays enter the parietal region about 2 inches posterior and  $1\frac{1}{2}$  inches superior to the external auditory meatus. This should project the optic

foramen near the center of the orbit. To the median side and below is the sphenoid sinus. Above the rim of the orbit is the frontal sinus. The ethmoids occupy the space below the frontal and extend posteriorly to the sphenoid.

For the frontal sinus alone the Caldwell position is ideal. If operative interference is anticipated this position should be supplemented with a lateral view, so that information concerning the depth of the sinus can be obtained.

For the ethmoids the oblique position of Rhese is best. These oblique positions give perspective views of all the sinuses from the frontal to the sphenoid. Both sides should be taken for comparison.

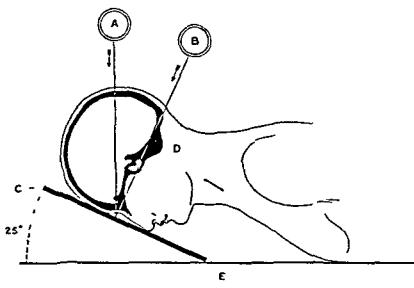


FIG. 120.—Schema showing the proper position for making a roentgenograph of the frontal and ethmoid sinuses. *A* the proper angle for passing the roentgen rays through the head *B* the improper angle as the rays must pass through a great deal of dense bone (*D*) to reach the sinus. *C* an 8-by-10-inch photographic film against which the forehead should rest. *E* the table upon which the patient lies. The forehead should be placed upon a triangular block with an inclination of 25 degrees as this is more comfortable to the patient and renders the line (*A*) perpendicular to the table.

If a picture of the antrums alone is wanted an excellent view is obtained from the extended chin position first described by Waters and Waldron in which the petrous portion of the temporal falls below the floor of the antrum. This position is obtained by having the chin of the patient touch the film and the tip of the nose from 1 to 1.5 cm. from the film. In a concave face an increase of 0.5 cm. in the distance of the nose from the film is required. This position gives a slight distortion of the frontal sinuses, due to the distance from the film. The posterior ethmoid cells are shown below the anterior group. This can be demonstrated by means of bismuth paste or metal clips.

A satisfactory view of the sphenoid is sometimes hard to get. Pfeiffer has described a method whereby the two sphenoids are shown side by

side. The patient extends the chin forward over the film and the central rays pass vertically through the head in the median line at a point 2 cm. in front of the external auditory canal. Bowen places the film on top of the extended head and passes the rays from below up. A combination of lateral, oblique and vertical views will usually give enough information to assist greatly in the diagnosis.

Beebe calls attention to the fact that the blurring or hazing of the sinus outline is of more diagnostic import than the shadow cast. He bases his objection to the roentgenogram taken stereoscopically on this fact.

**Radiopaque Oils**—Radiopaque oils injected into the sinuses are of distinct value in many cases of sinus diagnosis. These oils are composed of a bland vegetable oil with 40 per cent iodine content. They were first employed by Forrester in demarcating spinal cord lesions. They are of special value in the sphenoid and maxillary sinuses. By the use of the iodized oil the cavity of a sinus may be sharply outlined so that the thickness of the mucous membrane and the presence of polypi may be determined (fig. 121) and irregularities in size and shape can be accurately outlined. Diseased mucous membrane is shown by an unfilled zone between the oil mass and the bony outlines. It is also of value in aid in the detection of extensions of the sphenoid sinuses into the greater or lesser wings of the sphenoid bone.

The iodized oil may be introduced into the frontal sinuses by means of metal cannulas or small ureteral catheters and a syringe. The patient should be kept on his back on the table during the injection of the oil and not permitted to sit up until after the roentgenograms have been taken. The introduction of the oil can be observed under the fluoroscope from time to time to make sure that it is entering.

The sphenoid may be filled under the fluoroscope with the patient lying on his back. A long metal cannula with an appropriate curve is used to introduce the oil through the ostium. The oil may be injected into the sphenoid by puncture of the anterior wall if difficulty is found in injecting through the natural ostium.

**Technic**—With the head in the erect posture the needle is passed between the middle turbinate and the septum in a slightly oblique direction hugging the septum until the anterior wall of the sphenoid is reached. If there is insufficient space for the needle to pass the middle turbinate can be pushed or fractured laterally with a long nasal speculum or other suitable instrument. The needle held firmly against the sphenoid is raised to a horizontal plane parallel to the roof of the sphenoid. The cavity is entered at approximately the junction of the middle and lower thirds of the anterior wall.

The antrum is entered through the natural ostium by means of a Pierce antrum cannula. The same technic is employed as for irrigating except the radiopaque oil is left in the antrum until after the roentgenogram is taken.

The ethmoids are more difficult and frequently impossible, to fill except by the Proetz or Razer methods.

**Displacement Method** — About the same time Proetz<sup>1</sup> and Frazer<sup>2</sup> independently of each other introduced the displacement method in the diagnosis and treatment of sinusitis



FIG. 171 — Defects are filling with iodized oil of the left maxillary sinus. The left side of the face is next to the film and the oil has collected toward the zygoma. The irregular rivulets in the anterior and inferior portions outline the polyp in these areas. Numerous polyps were found at operation (Anderson Arch Otolaryngol.)

If for any reason the injection of radiopaque oil by means of a cannula is not advisable or possible Proetz's displacement method should be used. For the method to be effective (1) the ostia must be patent and

<sup>1</sup> Arch Otolaryngol 4:1 (July) 1926. Ann Otol Rhinol and Laryngol 36:297 (June) 1927.

<sup>2</sup> Monatschr f Ohrenh. 61:802 1927.

in normal relations to the sinuses, (2) the ostia must be covered by the fluid, (3) there must be negative pressure at the ostia

The method is especially adapted in filling the posterior series of sinuses. The frontal sinus is more difficult to fill by displacement. Obstructive lesions in the nose may prevent the proper filling of the sinuses.

Proetz's displacement method is accomplished by lowering the patient's head so that the chin and the external auditory meatuses are in the same vertical plane. While the patient says "K" to close the pharynx, 2 cc. of radiopaque oil warmed to body temperature are instilled into each nostril. The oil forms a pool in the upper portion of the nose into which the sinus ostia open. Intermittent negative pressure of 180 mm. of mercury is applied to one nostril, while the other is closed and the patient says "K". About ten negative pressure applications are usually required to suck the oil into the sinuses. Additional oil is now instilled and the procedure repeated. It is rare that more than 8 or 10 cc. of oil are needed. The patient is returned to the erect position and roentgen ray films taken to determine the degree of filling of the sinuses. Later films are taken at twenty-four-, forty-eight- or seventy-two-hour intervals to determine the emptying time. Normally the sinus should be empty by the end of ninety-six hours.

**Diagnostic Irrigations.**—In many instances a definite opinion concerning the presence or absence of pus or mucopus within a sinus cannot be formed without a diagnostic irrigation. This is done in the same manner as for therapeutic purposes either through the natural ostia or by puncture. Cultures or smears of the fluid may be taken

## CHAPTER XIII

### ETHMOIDITIS SPHENOIDITIS SINUSITIS IN CHILDREN AERO-SINUSITIS

HAVING considered the etiology, pathology and symptoms of sinusitis as a whole, a brief summary of ethmoiditis, sphenoiditis, sinusitis in infants and children and aëro-sinusitis will be given. A general discussion of sinusitis is not entirely satisfactory in considering these sinuses or in considering sinusitis as it occurs in infants and children.

#### ETHMOIDITIS

**Classification** — Involvement of the ethmoid may be acute or chronic, suppurative or non-suppurative. It may be classified also as (1) Acute congestive, (2) acute suppurative, (3) chronic hyperplastic and (4) chronic suppurative.

**Acute Congestive Ethmoiditis** — An acute congestive inflammation probably occurs in every case in which there is an acute inflammatory condition of the mucous membrane of the nose, as in acute rhinitis, etc. A chronic ethmoiditis or similar involvement in other sinuses is an important etiologic factor in the production of repeated attacks of colds. Systemic diseases, allergies, vitamin deficiencies, poor health habits and hygiene play important parts in the production of repeated attacks of acute ethmoiditis.

The symptoms of acute ethmoiditis are similar to those of acute rhinitis. The patient complains of a stuffy feeling in the nose with a profuse seromucous discharge and, as a rule, some sneezing. Headache between the eyes is a common complaint. Occasionally slight tenderness just back of the inner canthus of the eye is elicited.

**Acute Suppurative Ethmoiditis** — Acute suppurative ethmoiditis is not so common. It is frequently associated with an acute suppurative infection of an adjacent sinus. Sinusitis in infants and young children is largely confined to the ethmoid as it is the only sinus that has an early development. As a rule, one of the acute infectious diseases such as influenza, diphtheria, scarlet fever or measles precedes its onset. The mucous membrane is inflamed and covered with a thick purulent secretion.

The symptoms are occlusion of the nares, headache, with occasional neuralgic pains radiating to the back of the eye. Ocular symptoms may consist of tenderness of the bulb, painful rotation, orbital neuralgia and epiphora. Anosmia is present on the side of the nasal obstruction. The constitutional disturbances are slight fever, at times malaise, and the symptoms of a slight toxemia.

The prognosis of the acute form is good.

Treatment of acute ethmoiditis should be the reestablishment of ventilation and drainage. Between attacks, correction or elimination of



the systemic causative factors and obstructive lesions in the nose should be attended to. Heat administered with an infra-red lamp often gives marked relief. Tampons of 10 per cent mild silver protein in the middle meatus, left in place for thirty minutes, have a very beneficial action.

Surgery is rarely indicated in an acute attack, unless the disease has extended into other structures, such as the orbit. Fracture of the middle turbinate toward the septum is necessary in some cases. Turbectomy, as a rule, is not indicated in acute infections.

**Hyperplastic Ethmoiditis.**—Chronic catarrhal inflammation or hyperplastic ethmoiditis is very frequently seen.

The causative factors may be a nutritional disturbance such as protein sensitization or other forms of allergy and gastro-intestinal or endocrine disturbances. Nasal stenosis alone does not cause it.

In hyperplastic ethmoiditis an anaplasia of the nasal mucous membrane, especially in the region of the middle turbinates and ethmoid bulla, is observed. A degeneration of the mucous glands with infiltration of connective tissue occurs. At first the mucous glands are hypertrophied and may show a cystic dilatation of their acini. The hypertrophic process extends to the periosteum with fibrous degeneration along the bone. Mucous polyps frequently accompany this type of ethmoiditis. *This simple type of polyp seems to be due to an allergic condition in most instances.*

The symptoms are frequent or continuous head colds with sneezing and a serous nasal discharge. A granular hyperplastic pharyngitis is usually associated. Eustachian tube congestion, bronchitis and asthma are frequent accompaniments of this form of ethmoid disease. Scotomata, neuralgic pains around the orbit, photophobia and hyperemia of the conjunctiva may be present in some cases.

**Chronic Suppurative Ethmoiditis.**—Chronic suppurative ethmoiditis may occur as an open or as a closed form (empyema). Defective drainage is an important factor in the process as it is due to bacterial infection. It is usually associated with a disease of the other sinuses.

A round-cell infiltration with a proliferation of the epithelium, often absent in spots, is present. The denuded areas of the epithelium are replaced by granulation tissue.

The symptoms are variable, depending upon the virulence of the infection, the extent of the process and the general condition of the patient. Subjective discomfort such as a dull headache or pressure between the eyes, may be present. In a closed empyema pain over the root of the nose and vertex or a deep seated pain in or between the eyes may be present.

The exudate is purulent and shows a tendency to dry and form crusts. Remissions followed by acute exacerbations are common. The chronic hyperplastic form may be accompanied by suppuration if a secondary infection occurs.

The complications of chronic suppurative ethmoiditis are external fistula, rupture into the orbital cavity, especially in infants and children,

inflammation of the lacrimal duct cerebral complications otitis media focal infections and disturbances of vision

The local treatment consists of applications to the middle turbinate region of tampons soaked with 10 to 20 per cent aqueous solution of a mild colloid silver two or three times a week Medical diathermy Proetz's displacement irrigation vaccine therapy and non specific protein therapy may be of value Suction or negative pressure is particularly applicable to the ethmoid and frontal cells It should be intermittent in character and should be employed with caution and small force Suction through a fine tube at times is a useful adjunct Shrinking solutions such as a 1 to 3 per cent ephedrine applied to the region of the middle turbinate gives temporary relief

The removal of infected or enlarged tonsils and adenoid will cure many cases in children Hypertrophied or infected lymphoid structures should have attention Fracture of the middle turbinate or partial turbinectomy may be indicated if the ostia are blocked All polyps present should be removed Drainage of the bulla ethmoidalis by means of a punch forceps or a curette is sometimes successful

The intranasal or extranasal extenteration of the cells leaving the middle turbinate intact may be necessary if other means fail

### SPHENOIDITIS

Sphenoiditis may be acute or chronic congestive (catarrhal) or suppurative

**Acute Congestive Sphenoiditis** — This form may occur with any acute infectious process involving the nasal cavity or nasopharynx such as acute rhinitis the acute exanthemata etc The condition may go undiagnosed due to its mild character short duration or lack of symptoms

The symptoms may be a dull headache in the center of the head or occiput radiating to the vertex Reflex pain referred to the retro-orbital or periorbital regions the side of the head (parietal) or down the neck may be complained of for a day or so The congestion usually subsides without suppuration as the associated rhinitis or nasopharyngitis improves

The treatment is the same as for acute rhinitis The reflex pain to the orbital regions may be stopped in many cases by applying a 10 per cent cocaine solution to the sphenopalatine ganglion

**Acute Suppurative Sphenoiditis** — Acute sphenoid suppuration is characterized by headache neuralgic pains tightness in the head and vertigo The headaches may be the result of toxemia pressure within the sinus or possibly from an irritation of the Vidian nerve or sphenopalatine ganglion In the latter event the neuralgic headache is found in the eye radiating to the temple or mastoid region and to the nape of the neck The occipital pain may be the result of a Vidian nerve irritation also

A postnasal discharge with or without a crust formation is present as a rule Hacking or clearing the throat occurs several times a day

An associated hoarseness or aphonia anosmia a subjective sense of odor or parosmia may be present A dry productive cough frequently occurs

Mental symptoms such as somnolence insomnia or inability to concentrate may be complained of for a few days

Partial deafness or fulness in the ears with tinnitus and vertigo may be present if inflammation in the region of the eustachian tubes occurs Palmer<sup>1</sup> attributes some of the tinnitus and vertigo which so frequently accompanies a sphenoid infection to an irritation of the superior cervical sympathetic ganglion

Ocular manifestations may be redness pain asthenopia muscle imbalance photophobia and impaired vision (optic neuritis or atrophy etc)

The general symptoms may be gastric irritability respiratory disturbances fever increasing pulse rate and symptoms of toxemia

Examination shows pus between (above) the middle turbinate and the septum Mucus or pus may be seen in the nasopharynx occasionally with crusting The oropharynx may be dry and glistening or streaked with mucopus Nasal suction cautiously applied may demonstrate the pus that would otherwise not be seen Nasopharyngoscopy may be of much assistance in viewing the sphenoid area The pharynx may show a granular or a follicular pharyngitis with enlarged pharyngeal lymph nodes

Röntgenography of the sinus is of great help in the diagnosis The Granger position is made at an angle of 107 degrees in the antero-posterior position If the Granger position is used the G line becomes blurred or disappears in sphenoid suppuration The injection of a radiopaque oil may be of assistance in determining the presence or absence of thickening polyps cysts etc.

Irrigation of the sphenoid sinus determines the presence or absence of pus within the sinus If the pus reappears in the olfactory fissure after irrigation of the sinus its origin in the ethmoid labyrinth should be suspected

The local treatment consists in shrinking suction irrigations and such other measures that will promote free drainage and an increase of leukocytosis In some instances the fracture or removal of all or part of the middle turbinate is necessary The anterior sphenoid wall may be removed if necessary in the persistent or septic cases

Intracranial Complications

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If marked sepsis is present accompanied by a severe occipital headache and other signs and symptoms of a sphenoiditis a beginning intracranial complication should be suspected. The symptoms of meningitis such as photophobia, vertigo, persistent vomiting, meningeal cr. chills and stupor would demand immediate intervention if no other cause is found. A spheno-ethmoidectomy is usually performed in which the anterior wall of the sphenoid should be removed to the level of the floor and the sinus investigated for pathologic changes in the mucosa and bone. If osteomyelitis of the basilar sphenoid is present the mucosa of the sinus should be removed and the cancellous bone opened by means of a curette or chisel.

**Chronic Sphenoiditis** The chronically diseased sphenoid sinus may be without symptoms other than a postnasal discharge, headache at times and with or without symptoms of toxemia. At other times the various pre-symptomatic, mental, ocular, olfactory and toxic symptoms mentioned in the acute suppurative type may be present especially during an acute exacerbation.

The objective signs usually found are a congested mucous membrane in the postethmoid and sphenoid regions with a discharge at times in the olfactory fissure and on the pharyngeal wall. The pharyngeal discharge has usually a dark yellow or greenish color and clings tenaciously. A catarrhic odor may be present. Enlargement of the blind spot and paralysis of the external ocular muscles may occur in rare instances.

The sphenoid mucous membrane is thickened with some fibrosis, hyperplasia and occasionally polypoid changes and at times an osteitis.

The diagnosis is established from the symptoms, signs, roentgenograms and from demonstrating the pus or mucopus in the sphenoid cavity by a diagnostic irrigation.

**Treatment**—The general treatment consists in suitable regular exercise with the elimination of foods known to disagree with the patient and the selection of foods representing a proper balance of protein, fat, carbohydrates and vitamins. The intake of fluids sufficient for good elimination is indicated. During the acute exacerbations salicylates should be given to control headache if present. Physical therapy such as hot fomentations, radiotherapy or diathermy may be tried. Avoidance of insufflations of water while swimming or diving should be observed.

Local therapy such as tampons with colloidal silver salts, negative pressure and medicated steam inhalations may be of use.

Repeated irrigations of the sinus either through the natural ostium or by puncture of the anterior wall or by Proetz's displacement irrigation method are effective in many instances.

In other cases some form of intranasal surgery such as the removal of obstructive lesions or the removal of the anterior sphenoid wall is essential to effect a cure. If intracranial complications are suspected or in

some toxic cases some form of an external approach such as the sphenoidectomy may be necessary

### SINUSITIS IN CHILDREN

**Etiology**—Sinusitis in children usually is seen between the fourth and tenth years of age. Its frequency is about the same as in adults; however, many writers seem to think that sinus disease in children is largely regional in this country. Climatic conditions play a great part.

The ethmoid is well developed at birth and probably is involved more frequently than the other sinuses. The maxillary sinus is not fully developed until puberty. It rarely gives trouble before two years of age but may become infected from the day of birth. The sphenoid has clinical significance from the third year on but does not reach full development until the twelfth year. The frontal sinus may become involved from birth on but does not attain much size until the twelfth year. Involvement is rare under six years of age.

The predisposing factors are diminished resistance from any cause such as vitamin deficiency especially A, C and D; endocrine dyscrasia; metabolic disturbances; certain hereditary factors and poor hygienic or climatic conditions. Allergy is especially important in the etiology of sinusitis in children. Heredity is a factor as in adults. It frequently follows acute infections of the nose and throat such as acute rhinitis, tonsillitis and adenoiditis, scarlet fever, measles, influenza, pneumonia, diphtheria and whooping cough.

Local factors predispose to sinusitis such as an enlarged adenoid which interferes with nasal ventilation and drainage, foreign bodies and tumors in the nasal cavities, obstructive lesions and malformations.

The exciting cause is one or more of the various bacteria which may gain entrance into the sinuses from the nasal cavities through the ostia by extension along the mucous membrane. Extension may occur through the bony tissue as in osteomyelitis or in the maxillary sinus from an infected tooth root. It is possible to have an infection reach the sinuses by way of the blood stream and possibly through the lymphatics.

**Pathology**—The histopathology of chronic sinusitis in children is similar to that of the adult except the involvement in children is less extensive as a rule. Two general forms may be recognized: hyperplastic (polypoid, edematous, etc.) and atrophic (fibrotic, sclerotic). A mixed form may occur. The vasomotor or allergic sinusitis usually develops into the hyperplastic type in time.

In the hyperplastic form a general hyperplasia of the nasal mucosa and of the maxillary sinus is frequently found. Metaplasia of the epithelium is common, particularly if polypoid changes have occurred. Ulceration of the epithelial layer is rare. Loss of cilia or desquamation of the epithelium in the sinuses is not common. The subepithelial layer or basement membrane is rather faintly present in the sinuses but in chronic inflammations is greatly thickened. The hyperplastic form of sinusitis with polypi is very suggestive of allergic rhinitis.

The tunica propria or stroma consisting of connective tissue blood vessels and cellular elements (round polymorphonuclear eosinophiles and plasma cells) may show areas of dense infiltration particularly in the chronic polypoid type if suppuration is extensive. Cysts filled with purulent secretion are common. The tunica propria of the ethmoid has few secretory glands.

An edema with a polypoid or at times a cystic degeneration may occur. According to Spielberg<sup>1</sup> polypoid degenerations usually arise from the nasal mucosa lining the outer wall of the middle or superior meatus and rarely comes from the ethmoid mucosa.

Rarefaction, resorption or atrophy of the bone is frequently seen. It begins as an erosion of the ethmoid septa or the naso-antral wall. It is frequently associated with fibrosis and proliferative fibrous connective tissue changes. The bony atrophy is usually due to an endarteritis and vascular thrombosis causing a shutting off of the blood supply. Necrosis and hypertrophy of the bone are not so commonly seen. Necrosis may be found in chronic antrum suppuration involving the naso-antral wall. Bone hypertrophy is usually observed involving the inferior turbinate.

In atrophic sinusitis the epithelial layer of the nasal mucosa and turbinates usually shows dissolution, exfoliation or absence of the surface epithelium. The subepithelial layer is thickened as a rule. A round cell infiltration is usually present.

The tunica propria in atrophic sinusitis shows a low grade chronic inflammatory process with glandular atrophy. The periosteum may be thickened and infiltrated. The bone usually shows a retarded growth or underdevelopment with a sclerosis of the bony framework.

**Symptoms**—Sinusitis in children is characterized by a chronic nasal discharge from one or both sides of the nose. Frequent colds and ear aches occur as a rule. Examination frequently will disclose a malnourished and underweight child suffering from lack of appetite and constipation. The child may be inattentive and not do well in school.

Prolonged congestion, edema and nasal discharge are the usual manifestations. A pale edematous mucosa is usually associated with an allergic condition.

A discharge in the middle meatus in a child under six years of age usually means involvement of the ethmoid or maxillary sinuses as the frontal sinuses are not fully developed before this time and seldom become involved earlier. Discharge coming from above the middle turbinate before three years of age is indicative of a posterior ethmoiditis as the sphenoid sinus is not fully developed or does not often become infected before this age. Nasal discharge is most profuse on the side affected. It may be mucoid, mucopurulent or purulent.

Nasal obstruction on the involved side is usually present. It is more or less constant but may be intermittent.

Headache is a symptom of much diagnostic value in the acute cases in children over five years of age. Frontal headache most often indicates disease of the anterior group of cells. Occipital headache most

often indicates disease of the posterior group of cells. Morning frontal headache diminishing in intensity towards afternoon usually indicates a frontal involvement in an older child. Pain in the upper jaw or teeth increasing in intensity towards the afternoon usually indicates maxillary sinusitis. Tenderness to pressure over the thin areas of the sinus walls is of great assistance in the diagnosis in older children. These areas are the same as in the adult.

**Diagnosis**—The recognition of a sinus infection in children is frequently overlooked. This may be due to the fact that the roentgen ray and transillumination as a means of diagnosing sinus disease in early childhood are not so reliable as in adults. The diagnosis is also more difficult than in adults because subjective symptoms cannot be expressed. In older children indefinite pains around the head and face with headache may be complained of although these symptoms are not always present. Any chronic cold with a nasal discharge should arouse our suspicion of infection in these structures. Sneezing, headache, irritability and depression are important symptoms. The maxillary and especially the ethmoid sinuses chiefly concern us in children.

**Treatment**—General treatment consists in the proper regulation of diet and correction of nutritional disturbances. The use of foods containing the desired vitamins and minerals is necessary. In children the diet should contain liberal amounts of milk, cream, butter, eggs, vegetables and fruits. Cooperation with a good pediatrician is advisable.

Any allergic condition should be identified, the allergen eliminated if possible, otherwise a vaccine may be made from the offending allergen and administered.

Light therapy, ultra violet and infra red are of some value. The infra red rays stimulate phagocytosis and seem to attenuate heat sensitive microorganisms. A dilation of the blood vessels with an active hyperemia is produced with a beneficial effect.

**Internal Medication**—In acute cases small doses of atropine sulfate or in older children ephedrine sulfate is sometimes of use. Calcium with small doses of parathyroid often is beneficial. With evidence of thyroid deficiency, thyroid extract in small doses is given. Cod liver oil may be given once or twice daily. In subacute or chronic cases iodide of iron is of much value. Vaccines may be of aid in many cases. If much pain is present small doses of codeine combined with acetyl salicylic acid is comforting.

**Local treatments** consist of applying a small amount of 1 per cent ephedrine preferably in aqueous solution to the region of the middle turbinate two or three times a day. This may be followed by a small amount of suction cautiously applied for a short interval of one-half to one minute. Longer periods of negative pressure are seldom indicated in fact may be contraindicated due to a secondary edema that may follow.

During the painful stage of an acute sinusitis much comfort is derived from a small cotton tampon soaked in a 1 per cent ephedrine solution placed just beneath the middle turbinate and left in place for from two

to five minutes. In the late stage of an ethmoiditis tampons of mild silver protein placed in the middle meatus are of great value. These tampons should be left in place for from twenty to thirty minutes.

In older children the maxillary sinus may be irrigated through the natural ostium in most instances local anesthesia is sufficient. If the antrum trocar is passed through the inferior meatus it should be inserted upward and outward beneath the attachment of the inferior turbinate through the naso-atrial wall. The floor of the antrum is frequently above the inferior turbinate in infants and children.

Irrigating the affected sinus with a solution of penicillin (500 units per cc.) in 0.9 per cent saline or 1 per cent ephedrine solution has given excellent results. If penicillin is not available irrigating the sinus with a 1 to 20 per cent suspension of sulfathiazole microcrystals in normal saline should be done if sensitivity to sulfathiazole is not present.

**Surgical Procedures**—If used at all surgical procedures should be conservative. The establishment of ventilation and drainage with the least possible trauma is of the greatest importance.

If enlarged or infected tonsils and adenoid are present they should be removed. Dean and Armstrong have made a routine examination of the nasal sinuses of 1108 infants and children under fourteen years of age. They found a rather large number of chronic empyemas in their series most of which were apparently cured by the removal of diseased tonsils and adenoids. 80 per cent of their cases had this favorable termination.

Operative procedures on the sinuses themselves in children are not often indicated. The acute infections of these cavities usually disappearing of themselves. Those cases which are classed as chronic empyemas yield much more readily than in the adult to non-operative procedures due to two things: (1) The age of the patient excludes long chronicity. (2) nasal obstructive lesions at this age are not common.

If the maxillary infection does not clear up after a thorough trial by conservative treatment additional ventilation and drainage may be obtained by making an opening beneath the lower turbinate. Such openings usually close rapidly in children.

Ether is the anesthetic of choice because the operative procedures can be deliberate and thorough. In those cases with kidney trouble either a nitrous oxide or an ethylene mixture is safer than ether.

A suitable antrum trocar is inserted under the inferior turbinate and the nasal wall of the antrum is pierced in an upward and outward direction.

The opening is enlarged with a rasp or small biting forceps to permit the introduction of a rubber catheter. The catheter should extend from the interior of the antrum into the vestibule of the nose.

Irrigations or instillations are made through the tube.

The tube is removed on the fifth or sixth day. Subsequent irrigations are made with a straight needle or a curved trocar.

**Complications**—The complications of sinus infection in children may be pyelitis, gastro-intestinal disturbances, cervical adenitis, meningitis.



brain abscess except in infants (Dein) otitis media and orbital complications such as orbital cellulitis retrobulbar neuritis etc

### AVIATION SINUSITIS AERO-SINUSITIS

Acute inflammation of the lining mucous membrane of the sinuses hemorrhage into the sinuses or even sinusitis may result from rapid barometric changes in altitudinal flights. These pathologic changes may occur if the ostia of the sinuses are closed by redundant tissue or are covered by a purulent secretion. During ascent the air within the sinuses is reduced in conformity with the changed barometric pressure but during descent redundant tissue may be sucked within the sinus ostium and act as a ball and flutter valve. The reduced air pressure within the sinus thus temporarily created may result in an acute swelling inflammation or bleeding of the mucosa. Acute pain in the region of the affected sinus is usually experienced until the air equilibrium is restored.

Infected mucus or pus over the sinus ostium may be sucked into the sinus during descent resulting at times in an acute sinusitis.

Shrinking the redundant tissue in the regions of the sinus ostia before the flight is begun may prevent these mishaps from occurring. Otherwise they should be treated as for an acute sinusitis. Patients with an upper respiratory infection should not fly.

## CHAPTER XIV

### THE GENERAL AND LOCAL TREATMENT OF SINUS INFECTIONS

The non-surgical treatment of sinusitis depends upon increasing the patient's resistance to the infection by the various means available, the establishment of free drainage and ventilation of the nose and sinuses and the removal of the infected secretions as they form.

To increase the patient's resistance an adequate balanced diet with the proper mineral and vitamin content is essential. Sufficient rest, good hygiene and regular habits are most advisable. If the infection is acute, recourse to penicillin and the sulfonamides used locally or parenterally would be an important help. It may be that streptomycin may play an increasingly important part.

**Shrinking and Ventilation** — To promote drainage and ventilation of the sinuses the ostia must be kept patent. This is most satisfactorily accomplished by applying a 1 to 3 per cent isotonic solution of ephedrine or one of the allied compounds to the region of the sinus ostium. If the anterior group of sinuses are involved a pledget or tampon of cotton moistened with the shrinking solution is placed in the middle meatus beneath the middle turbinate and left in place for from five to ten minutes. After the ephedrine tampon has been removed gentle suction may be made as described later in this Chapter. If the sinus is very painful a 0.5 to 1 per cent solution of cocaine may be used instead of the ephedrine.

The hyperplastic form of sinusitis in which some allergen is the causative factor should have the treatment directed to the cause rather than to attempt some surgical procedure before removing or treating the agents producing the pathology.

**The Sulfonamides and Penicillin** — The sulfonamides and penicillin are less efficacious in the treatment of sinusitis than in many diseases. They are most helpful in the early acute stage. If any evidence of an accompanying cellulitis or other complication is present they should be administered in full doses. Acute ethmoiditis responds more readily than do infections of the other sinuses. A chronically infected sinus is not helped much by the sulfonamides or by penicillin. Chemotherapy should not be used to the exclusion of other established means.

For local use especially for instillation after irrigation penicillin 500 to 1000 units per cc. seems to be more efficacious than the sulfonamides. However a 5 to 20 per cent suspension of sulfathiazole microcrystals in normal saline instilled into an acutely infected sinus after a preliminary irrigation has given excellent results.

**Physical Therapy** — (Chap. LX.) *Heat* dry or moist or the heat from an incandescent or infra red lamp applied over the face sometimes affords speedy relief. The lamp should be applied over the closed eyes,

at a distance of from 12 to 18 inches for twenty to thirty minutes. The good effects are due to the increased hyperemia and leukocytosis and to the improvement of the nutrition.

*Diathermy* may be used in chronic sinusitis but should be avoided in acute empyema. If used it should be applied over the affected sinus in the form of a small metal plate about the size of the sinus and another larger plate on the back of the neck. The current should be used up to the tolerance of the individual without burning the skin. Thus if used from five to ten minutes will give relief in some cases.

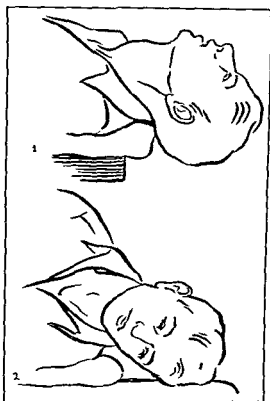


FIG 129.—Two head low positions for instilling drops in the region of the various sinuses. 1 The Froetz position, indicated when the ostia of the posterior group of sinuses are to be reached. 2 The side position will permit the drop to reach the ostia of all the sinuses if the head is rotated slowly back and forth.

*Röntgen ray* therapy of acute sinusitis seems to give relief of pain and headache in most instances. Small doses should be given early in the disease.

*Quartz lamp or ultra violet light* is of value in the later stages. The air-cooled lamp may be used for its general physiologic action. The water-cooled quartz lamp with quartz nasal applicators is used for the local action on the nasal mucosa.

The treatment of chronic sinusitis is a more difficult type to treat

successfully on account of its chronicity which of itself may imply that anatomic barriers existed during the acute stage which prevented resolution. These barriers if present must be overcome before a cure can be established. The anatomic barriers to resolutions may consist of hyperplastic changes in the mucous membrane of the nose especially in the region of the cell openings and the olfactory fissure or they may be due to ethmoid cells in the middle turbinate deviations of the upper portion of the nasal septum polypoid tumors etc.

The swelling of the mucosa may be somewhat reduced by the local application of ephedrine or cocaine. Suction followed by swabbing the nose with 1 to 2 per cent silver nitrate solution or 10 to 20 per cent mild silver protein is many times effective.

Gampons of mild silver protein placed just beneath the middle turbinate are of especial value in treating the subacute or chronic forms of ethmoiditis.

### IRRIGATIONS OF THE SINUSES

In the simpler form of sinusitis that is when there are no granulations or necrotic bone the persistent irrigation of the affected sinus with an isotonic or mild antiseptic solution followed by the instillation of a penicillin solution or a sulfonamide suspension is frequently followed by a cure. The recently advocated use of a 5 per cent solution of sodium sulfathiazole (Turnbull) for sinus or nasal irrigations should be discouraged as it has an injurious action on the sinus and nasal mucous membranes probably due to its caustic action (pH 8 to 10).

Sulfathiazole powder blown into the sinus through an irrigating cannula or a 20 to 50 per cent suspension in a lubricating jelly (Marks\*) injected into the sinus are effective in many instances.



Fig. 124. Irrigating cannula.

**Irrigation of the Frontal Sinus** — The irrigation of the frontal sinus may be performed through the fronto-nasal canal except in those few cases in which it is closed by an enlarged bulla or by an enlarged middle turbinate. An understanding of certain anatomic peculiarities of the region of the infundibulum and the fronto-nasal canal will aid materially in the irrigation of the sinuses.

The hiatus semilunaris is a slit like crescentic-shaped opening in the outer wall of the nose. It is the opening of the infundibulum into the middle meatus. Its inner lip is the upper margin of the uncinate process of the ethmoid bone.

The infundibulum is a deep narrow groove or gutter in the outer wall of the nose (Fig. 124 f) the inner wall of which is the uncinate process. The fronto-nasal canal drains into the infundibulum in about one-half

of the subjects whereas in the remainder it drains a little anterior to it directly into the middle meatus.

The fronto-nasal canal is a closed tubular duct extending upward and forward from the middle meatus or the infundibulum as the case may be, to the frontal sinus. Its opening into the floor of the frontal sinus is known as the ostium frontale. In rare instances the ostium opens high upon the posterior wall of the sinus.

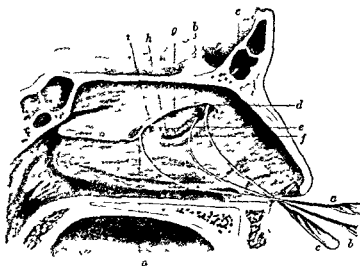


FIG. 124.—Probing (or irrigating) the frontal sinus. The anterior half of the middle turbinate is removed to show the anatomic landmarks: *a a* the probe in the first position beneath the middle turbinate and posterior to the bulla ethmoidalis; *b b* the probe in the second position beneath the middle turbinate and in front of the bulla ethmoidalis; *c c* the probe in the third position introduced through the fronto-nasal canal into the frontal sinus; *d* the nasal end of the fronto-nasal canal; *e* the lip of the uncinate process; *f* the inner wall (uncinate process) of the infundibulum; *g* the ostium bulla ethmoidalis; *h* the ostium maxillare; *i* an accessory opening into the maxillary sinus (drawing from a specimen loaned by Dr. Ira Frank).

The hiatus semilunaris is the key to the insertion of the cannula, as it is the opening into the infundibulum, which must be entered to reach the fronto-nasal canal in about one-half of the cases. The bulla ethmoidalis is situated just above the hiatus, and when large it encroaches upon the slit-like opening and partially or completely closes it. Occasionally there are accessory cells in the uncinate process, which also obstruct the hiatus. In other cases the middle turbinate closely hugs the outer wall of the nose and blocks the hiatus. When either of these anatomic peculiarities is present the introduction of the probe or the cannula is rendered difficult or impossible. If the fronto-nasal canal opens in front of the infundibulum the cannula may be passed into it even though the hiatus is closed.

Another difficulty sometimes encountered is, that the cannula may enter the ostium of one of the anterior ethmoid cells instead of the frontal sinus. Some of the anterior cells may open into the infundibulum on its outer wall, while others open into the fronto-nasal canal. The ante-

rior cells are usually located external to the infundibulum and the fronto-nasal canal and their ostia open into the infundibulum and fronto-nasal canal through the outer wall. In inserting the cannula therefore the point of the cannula should be kept against the inner or mesial wall of the fronto-nasal canal in order to avoid the ostia on its outer wall.

Irrigation is generally more difficult in those subjects in which the fronto-nasal canal empties into the infundibulum than when it empties directly into the middle meatus. In the former case the canal is often tortuous and narrow while in the latter it is usually straighter and of larger caliber.

The middle turbinate is sometimes so close to the hiatus especially when the turbinate contains an accessory cell that it is difficult to enter it with a probe or cannula. In this event the removal of the anterior third of the middle turbinate overcomes the difficulty.

**Technic of Irrigating the Frontal Sinuses**—First ocannize the parts then introduce a fine silver cannula (Fig. 123) bent at its distal end to an angle of about 135 degrees between the anterior third of the middle turbinate and the outer wall of the nose. Keep the tip of the



FIG. 123. Holmes' malloale frontal sinus probe

cannula against the outer surface of the turbinate and pass it forward and upward through the hiatus into the infundibulum where it readily enters the fronto-nasal canal even to the frontal ostium (Fig. 124).

After engaging in the middle meatus it should be passed into the infundibulum and canal for about 6 to 8 cm. to reach the frontal sinus.

The syringe is attached to the cannula and the sinus gently irrigated with warm normal salt or boric acid solution.

**Irrigation of the Maxillary Sinus Through the Ostium** This can be effected through the normal antral opening in most cases by means of Lierce's antrum cannula.

**Technic**—After anesthetizing beneath the middle portion of the middle turbinate the cannula is introduced upward and backward with the curved tip in a vertical position. When the anesthetized portion of the middle turbinate is reached the tip of the cannula is inserted beneath the middle turbinate in an upward and outward direction so as to pass over the uncinate process. As this process is passed over the tip of the cannula is directed downward and outward (sometimes directly outward) when it will usually enter the natural opening. In a few cases it is impossible to irrigate by this method on account of the hidden position in the infundibulum of the naso-antral opening and on account of its forward and downward direction from the infundibulum to the antrum. The opening into the antrum in these cases is not directly through the

lateral wall of the nose but it is more like a canal extending obliquely downward and forward through the thickness of the wall. In a certain number of cases there are accessory openings into the antrum (Fig. 124) which when present may be utilized for purposes of irrigation.

**Irrigation of the Maxillary Sinus by Puncture Through the Inferior Meatus**—If difficulty or an excessive irritation of the tissues are encountered from irrigating through the natural opening, an artificial route should be chosen. The most available one being the inferior meatus. A curved or straight trocar being used for the purpose.

**Technic**—Anesthetize the mucous membrane of the naso-antral wall of the inferior meatus with a 5 per cent solution of cocaine.



FIG. 126 Pierce's antrum cannula for irrigating through the natural opening.

Introduce the trocar beneath the inferior turbinate posterior to the anterior antral wall and direct it upward and outward a little above the floor of the nose in order to avoid the thick wall of bone at this point. In some cases, especially when a maxillary cyst is present or in infants and children, the floor of the antrum is quite high and it is not possible to introduce the trocar beneath the inferior turbinate. In this event puncture beneath the middle turbinate through the membranous portion of the naso-antral wall can be done.



FIG. 127 Charlton's antrum trocar.

The dangers of antrum irrigation by puncture are largely a matter of faulty technic. The trocar should have a very sharp point to avoid pushing the lining membrane before it after piercing the bony wall. Secondly, there should be an avoidance of any undue force in using the irrigating fluid.

After penetrating the naso-antral wall, remove the trocar, leaving the cannula in position. Attach the rubber hose of the syringe to the cannula and irrigate with normal salt or other solution chosen for the purpose.

The irrigations may be repeated every three or four days as long as necessary through the artificial opening.

**Irrigation of the Maxillary Sinus Through the Alveolar Process**—This method is mentioned only to be condemned unless the alveolar

opening is permitted to close before the epithelium has extended into it otherwise a chronic fistula results with constant reinfection of the antrum. It is applicable only to those cases of antrum infections secondary to a root infection of a tooth in which the abscess has eroded a fistula through the floor of the antrum.

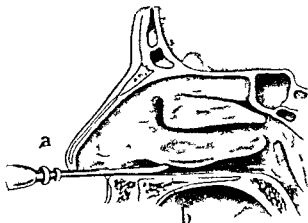


FIG. 198 — Introducing a trocar (a) and cannula into the maxillary antrum beneath the inferior turbinate near the junction (b) of the anterior and middle turbinates.

**Technic** — Select a place where a tooth has been extracted below the antrum or if a tooth is decayed beyond repair extract it for the purpose and drill a canal into the floor of the sinus (Cooper's operation). Through this opening a cannula is introduced and the antrum irrigated with normal salt or any solution desired.

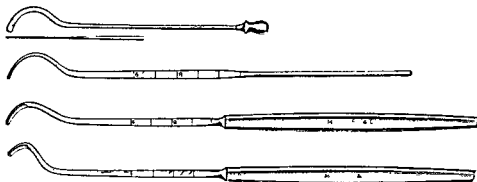


FIG. 199 — Andrews' sphenoid probe cannula and knives.

The canal thus made may be kept open for a short time only by means of a hard or soft rubber or gold tube made for the purpose. The tube should be flanged on the lower end to prevent it slipping upward into the antrum. A plug should be introduced into the tube to prevent the entrance of food into the antrum.

**Irrigation of the Ethmoid Cells** — This is often impossible except in the case of anterior cells which drain into the fronto-nasal canal. The



*bullae ethmoidalis* one of the anterior cells does not drain into the fronto-nasal canal but drains directly into the middle meatus and its ostium is situated at its upper median wall beneath the attachment of the middle turbinate.

The technic for the irrigation of the anterior cells opening into the fronto nasal canal is the same as for the frontal sinus this being introduced into the canal only to the second position (Fig 124) indeed both sets of cells are often irrigated at the same time. Their ostia are bathed with the irrigating fluid and the accumulated pus in the canal is removed thus facilitating the drainage of the cells.

**Irrigation of the Sphenoid Sinus Through the Ostium** — This is possible when the middle turbinate or a deflection of the septum does

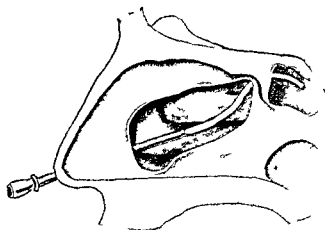


FIG 130 — Irrigation of the sphenoid sinus with curved cannula.

not prevent the introduction of the sphenoidal cannula into its opening. When such an obstruction is present it may become necessary to first remove it by some surgical procedure before the irrigations can be practised. A silver eustachian catheter may be used in place of a sphenoid cannula. The curve used for the inflation of the ear is the correct one for irrigation of the sphenoid sinus. Myle's cannula may be bent to reach any sinus and is smaller than the eustachian catheter.

When the cannula has been introduced the patient should be instructed to lean forward and open his mouth, then the hose of the syringe should be attached to the cannula and the sinus irrigated. If the patient's head is inclined forward and the mouth open the fluid will not enter the eustachian tube.

**Irrigation of the Sphenoid Sinus by Puncture** — Puncture of the anterior wall may be done if the natural ostium is not accessible.

After local anesthesia a long bladed Killian speculum is inserted between the septum and the middle turbinate separating the two. The anterior wall of the sphenoid can now be seen. A sphenoid trocar is introduced backward and upward until the sphenoid wall is reached.

The handle of the trocar is now raised. By pushing it backward the wall is punctured. The opening can be enlarged with a biting forceps or a rasp if necessary.

Should granulations be abundant it may be necessary first to remove the middle turbinate and then the anterior wall of the sphenoid sinus and cautiously curette its interior.

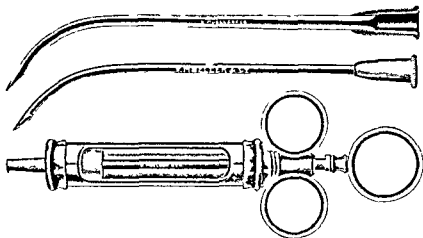


FIG. 131—Walsh's antrum exploring syringe

Irrigation of the sinuses in chronic suppurative inflammation is upon the whole an unsatisfactory therapeutic measure, however, before resorting to surgical means it should be given a thorough trial.

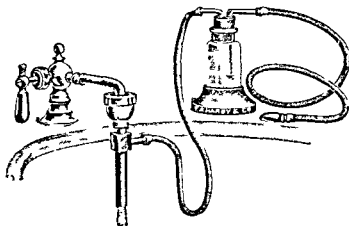


FIG. 132—Brawley's vacuum aspirator

Irrigation is more useful in the simple suppurative cases uncomplicated by granulations and necrosis. The removal of the purulent secretions gives the irritated epithelium a chance to regenerate. If after a few weeks' trial the case does not greatly improve, irrigation should be discontinued and some other method of treatment, probably surgical in character, instituted.

**Displacement Irrigations** — In many cases of acute sinusitis that have not responded well to other treatments Proetz's displacement irrigations (described elsewhere) may be tried with good results in most instances. The solutions used are physiologic salt  $\frac{1}{2}$  to 1 per cent ephedrine or penicillin (500 units per cc). Displacement irrigations should not be done during the first three or four days of the acute infection as a secondary hyperemia of the mucosa and sinus ostium frequently follows. Treatments may be done one or two times daily for three or four days.

### TREATMENT BY NEGATIVE AIR PRESSURE

The *rationale* of this method of treatment consists chiefly in the increased hyperemia of the mucous membrane lining the cells and the mechanical removal of secretions. The local nutrition is thereby improved, the cell resistance and leukocytosis increased and the infective process checked.

**Technic** — The apparatus necessary for producing negative pressure in the sinuses consists of either a hand pump or other device for exhausting the air in the nasal chambers. Brawley's apparatus is operated by attaching it to a faucet of the wash basin, the negative pressure being regulated by the amount of water turned on. The electric motor driven pumps are more convenient.

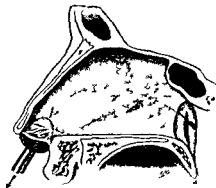


FIG. 133 — Showing the soft palate closed during suction through the nose

Insert the nasal tip into one nostril, holding the other nostril closed by means of a finger and bring the soft palate into apposition with the pharyngeal wall by swallowing or saying *kick kick*. With practice the patient soon learns to close the palate without difficulty.

While the air is thus exhausted the pus is drawn from the sinus into the nasal fossa or into the rubber tubing. In this way considerable pus may be removed. Excessive or too long-continued suction should be avoided.

Daily sittings should be maintained until improvement begins or until the surgeon is convinced that this method of treatment is inadequate for the case.

Drainage is facilitated if the head is turned so as to bring the ostium of the affected sinus to the most dependent position.

## CHAPTER XX

### III. SURGICAL TREATMENT OF SINUSITIS

#### THE INDICATIONS FOR SURGERY OF THE SINUSES

CONSERVATIVE sinus surgery consists in aeration and drainage with little or no destruction of the sinus. In other words the preservation or the restoration of diseased parts rather than their removal or exenteration. Radical surgery involves destruction of part or all of the sinus or the removal of large parts of the bony wall or of the lining mucous membrane.

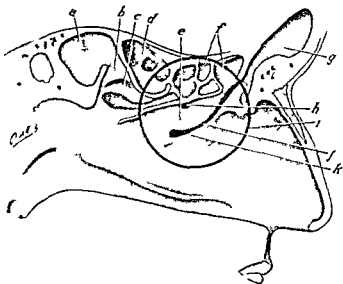


FIG. 134. The circle encloses the important structures concerned with the drainage and ventilation of the sinuses of the anterior group: *a* the sphenoid sinus; *b* the sphenoidal fossa; *c* the superior turbinate; *d* posterior ethmoid cells; *e* bulla ethmoidalis; *f* anterior ethmoid cells draining into the fronto-nasal canal; *g* frontal sinus; *h* the osium of the bulla ethmoidalis; *i* hiatus semilunaris; *j* the uncinate process or outer wall of the infundibulum or gutter on the outer wall of the nose into which the frontal, anterior ethmoid and maxillary sinuses usually drain. The highlight below and anterior to *j* and *k* indicates the inferior boundary of the infundibulum or gutter into which the sinuses drain. The middle turbinate has been removed to exhibit the anatomical details beneath it.

Under the first heading of conservative surgery would be the correction of obstructive septal deformities, removal of polypoid hyperplasias, removal or fracture away from the ostia of an obstructing middle turbinate, drainage of individual ethmoid cells rather than total exenteration and the making of a window in the naso-antral wall of the maxillary sinus rather than the more radical external approach.

A radical operation would be an external approach with removal of the lining mucous membrane or such intranasal operation as a complete intranasal ethmoidectomy.

Relative indications for radical sinus surgery are the continuance of the disability caused by the sinusitis continued and persistent headache or pain profuse and persistent discharge nasal crusting obnoxious odor due to the discharge or general ill health dependent upon sinus disease Nephritis arthritis carditis chronic cough caused by enlarged mediastinal lymph nodes bronchiectasis certain allergies (bacterial) gastrointestinal disturbances malnutrition malaise and other general conditions may be attributed to sinus infection

Positive indications for radical surgery are a beginning intracranial extension such as meningitis or subdural abscess necrosis of the walls of the sinuses as shown by a fistula formation tumors of the sinuses such as malignant growths osteomas or fibromas or cavernous sinus thrombosis due to sinus disease progressive osteomyelitis of the skull dentigerous cysts of the maxillary sinus or mucocele of the sinus some cases of orbital cellulitis with pus formation and certain eye disorders associated with sinusitis such as retrobulbar neuritis when other causes especially multiple neuritis are not found

### INTRANASAL SURGERY OF THE FRONTAL SINUS

**Enlargement of the Fronto nasal Canal** — The intranasal enlargement of the fronto nasal canal is indicated when a mechanical blocking of the fronto nasal duct results from an encroaching ethmoid cell. In this event the secretions from an infected frontal sinus may not have adequate drainage. The operation is not performed until all acute symptoms have subsided.

The technic is the same as that described for Mosher's Intranasal Ethmoid Operation (described elsewhere) except the posterior ethmoid cells are not excised. Great care should be taken to avoid injury to the interior floor of the skull especially the delicate cribriform plate.

### EXTERNAL SURGERY OF THE FRONTAL SINUS

**Indications** — There are a certain group of cases in which some form of external procedure is indicated namely those cases with external fistula tumors intracranial complications bone necrosis and severe orbital complications. The various external frontal sinus operations such as the Hajek Luc Kuhnt Lothrop-Skillern Killian etc have been supplanted to a large extent by the Lynch type of operation as modified by Ferris Smith and Sewell. This procedure not only gives access to the frontal sinus through its floor but to the anterior and posterior ethmoid sinuses and if necessary to the sphenoid sinus.

**The External Fronto ethmo sphenoidal Operation** This external approach for a combined operation on the frontal ethmoid and sphenoid sinuses as described by Lynch<sup>1</sup> and modified by Ferris Smith<sup>2</sup> and Sewell<sup>3,4</sup> has supplanted older and somewhat similar procedures

<sup>1</sup> *Ann. Otol. Rhinol. and Laryngol.* 38: 931 (December) 1929

<sup>2</sup> *Arch. Otolaryngol.* 19: 157 (February) 1934

<sup>3</sup> *Arch. Otolaryngol.* 4: 37 (November) 1926

<sup>4</sup> *Surg., Gynec. and Obst.* 60: 588 (February 15) 1935

This type of operation is necessary in only a limited number of cases of severe intractable or otherwise complicated sinusitis as discussed under the Indications for Surgery of the Sinuses. If a radical extirpation of the sinuses is indicated the external approach is safer and easier than an intranasal one. The disadvantage of the external operation is the scar.

**Preparation of the Skin**—Thorough washing of the entire face with green soap and water followed by sponging with alcohol and then ether is usually sufficient. Some operators irrigate the nasal cavity with an alkaline solution and then paint the nasal mucous membrane with 2 per cent picric acid in alcohol. Most do nothing to the interior of the nose thereby avoiding injury and irritation to the mucosa. The eyebrow is not shaved. The lids are sutured or otherwise held together.

**Anesthesia**—Local or general anesthetic may be used. The steps in local anesthesia are as follows:

1. A sleeping sedative is given the night before.
2. 0.1 gm ( $1\frac{1}{2}$  gr) of pentobarbital or a similar barbiturate is given one and a half hours before operation and 0.012 gm ( $\frac{1}{8}$  gr) of morphine sulfate in 10.0003 gm ( $1\frac{1}{36}$  gr) of scopolamine sulfate are given forty-five minutes before operation.
3. The nasal cavity on the side to be operated is packed with gauze or cotton moistened with a 10 per cent solution of cocaine to which a few drops of 1 to 1000 solution of epinephrine have been added. Instead of packing the nose with cocaine-epinephrine solution the nasal mucosa may be swabbed with a small amount of cocaine flakes. A cotton mounted applicator is moistened with a 1 to 1000 epinephrine solution then dipped into the cocaine flakes and one to two applications given.
4. The proposed line of incision is infiltrated with a 1 or 2 per cent procaine solution with 6 to 8 drops of 1 to 1000 epinephrine added to the ounce of cocaine solution. A portion of the solution is infiltrated around the region of the supraorbital nerve.
5. A 21 gauge  $2\frac{1}{2}$  inch needle is inserted along the bone above the inner canthus of the eye following a line formed by the junction of the medial and superior walls of the orbit. The needle is inserted about  $1\frac{1}{2}$  inches to the region of the anterior ethmoid vessels where from  $2\frac{1}{2}$  to 3 cc of the cocaine-epinephrine solution is injected.

**Incision**—The incision (Lorris Smith) is made through the skin and periosteum. It begins at the orbital rim just below the eyebrow, is brought down around the inner canthus of the eye and about  $\frac{1}{4}$  inch away from it onto the nose and terminates 1 cm. below the inner canthus.

**Elevation of the Periosteum and Periorbita**—The periosteum is elevated from the floor of the frontal sinus (not the anterior wall) inferior to the incision and within the orbit. The elevation is usually begun most easily at the junction of the superior and medial orbital walls. It is then carried laterally until the floor of the frontal sinus has been uncovered. The elevation is extended inferiorly to display the lacrimal fossa and

posteriorly to within a few mm. of the optic foramen. Care should be taken to avoid buttonholing the periosteum. After the lacrimal sac has been displaced laterally the cribriform lacrimal bone is seen and posterior to this the lamina papyracea. As a rule the area of the superior oblique muscle must be dislodged.

The anterior ethmoidal vessels and nerves are encountered during the elevation of the periosteum from the medial orbital wall (lamina papyracea). The vessels rarely bleed much when severed but at times it is necessary to double ligate and divide them. The posterior ethmoidal vessels found still further posteriorly along the medial orbital wall are larger and after being anesthetized with a 10 per cent cocaine pack should be double ligated and divided.

**Opening Into the Sinuses**—Entrance into the ethmoid sinus is accomplished by means of some sharp instrument pressing against the posterior crest of the lacrimal fossa or the lacrimal bone. After an opening has been made through the bone up to but not through the nasal mucous membrane various sized Kerrison forceps and rongeurs are used to enlarge the opening. The Ferris Smith retractor aids in exposing the structures. The retractor is inserted so that the blades retract the periorbita while the teeth engage the periosteum. As the bone is removed superiorly just behind the nasal process of the frontal bone the frontal sinus is entered. Working posteriorly the anterior ethmoid cells are encountered.

The operation should be relatively bloodless so that the operator can under direct vision remove the entire floor of the frontal sinus including the lateral angle and all intrasinus septa. The entire mucous membrane of the frontal sinus is removed by means of variously shaped curettes. All the anterior and posterior ethmoid cells are also removed however in some instances the ramifications of the cells may make this difficult if not impossible. The nasal mucous membrane which has been preserved can be cut along the superior and anterior borders and used as a flap over the periorbita. The lamina papyracea is removed and with it the middle turbinate if it still remains.

The position of the cribriform plate must be kept in mind at all times. It is not easily fractured if care is exercised. A greater danger is the creation of minute openings into the subdural space by injury of the olfactory filaments through pulling or laceration. Lunch marks the position of the cribriform plate by means of a probe inserted through the nostril with the tip of the probe in contact with the plate.

As the posterior extremity of the ethmoid labyrinth is reached the anterior wall of the sphenoid sinus is seen. The natural ostium is identified and enlarged and the anterior wall removed by means of biting forceps or curettes. It may be necessary to ligate the sphenopalatine artery if anything more than enlargement of the antral ostium is done. Ligation of the artery is done by first elevating the periosteum. Then the artery together with the periosteum is tied. Lunch recommends removal of the anterior sphenoidal wall and all the sphenoidal mucous membrane. Ferris Smith removes the sphenoidal floor as well.

**Grafts and Flaps**—Lorris Smith<sup>1</sup> in his fronto-ethmoid sphenoidal sinus operation covers the exposed periorbital and cut edge of the nasal process of the maxilla with an epithelial graft applied over an inflatable rubber pad which furnishes the required pressure for the adhesion of the graft. The projection on the bag is inserted into the infundibulum of the frontal sinus. The flat surface is lubricated and covered with split skin with the raw surface exposed. This is approximated to the periorbita in the area of the removed lamina papyracea. This prevents adhesion of the periorbita to the cut surfaces of the bone and consequently it prevents obstruction of frontal ventilation and drainage. The bag may be used repeatedly. Sewall uses flaps prepared from the nasal mucosa to line the operative field and to prevent the closure of the nasofrontal opening. The periorbital flap may be sutured with catgut to the posterior margin.

**Drainage** Drainage by means of a cigarette drain or a soft rubber tube through the nose for two or three days is advisable. Irrigations of the operated area with solutions of penicillin can be carried out through the tube.

**Closure of the Wound**—The skin incision is closed completely with interrupted skin sutures. The eye is covered with pads for twenty-four hours.

A few cases have diplopia lasting for a few days.

Nasal crusting is relieved by tampons of a 10 per cent solution of mild silver protein.

## INTRANASAL SURGERY OF THE MAXILLARY SINUS

Puncture and irrigation by means of an antrum trocar are usually sufficient to effect a cure in acute and subacute inflammation of the sinus. The puncture may be made beneath the inferior turbinate. The cannula is introduced two or three times a week under cocaine anesthesia with little discomfort to the patient. The irrigating solutions usually used are normal salt, penicillin solutions and sulfathiazole suspensions as listed under General and Local Treatment of Sinusitis. If desired the antral irrigations may be attempted through the natural ostium by means of the Pierce antrum cannula.

If however the mucous membrane has undergone marked degenerative changes with involvement of the underlying bone it may be necessary to remove the naso-antral wall or to perform an extranasal operation such as the Caldwell-Luc.

**Removal of the Naso-antral Wall (Antrum Window)**—This operation was performed by Miles and has had many advocates since then. Clinical experience has shown that a small opening in the naso-antral wall quickly closes whereas a large one may remain open permanently.

The antral wall may be removed in the inferior meatus beneath the inferior turbinate or the antral wall may be removed in the middle



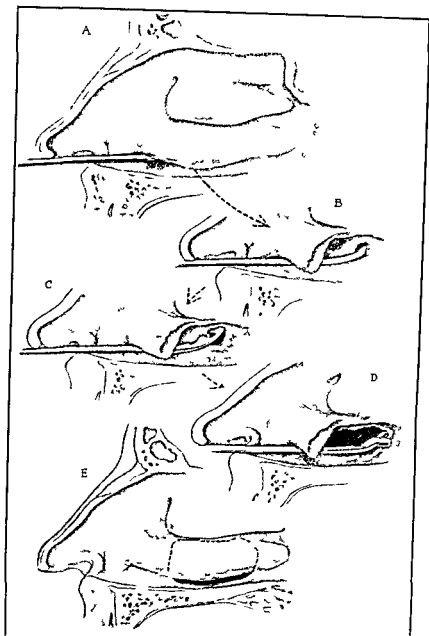


FIG. 13a.—Removing the naso-antral wall (antrum window) beneath the inferior turbinate. *A* Puncturing the naso-antral wall with a sharp pointed rasp or trocar. The hole should be made large enough to admit the bone-cutting forceps. *B* Enlarging the puncture by means of a curved rasp so as to admit the cutting forceps. *C* The forward cutting forceps are inserted and the naso-antral wall removed as far forward as possible. *D* The backward cutting forceps removing the posterior portion of the naso-antral wall. *E* The naso-antral wall removed beneath the inferior turbinate at the completion of the antrum window operation.

meatus beneath the middle turbinate. The usual procedure is through the inferior meatus

Many instruments have been devised for the removal of the naso-antral wall, some of which enable the operator to do the work with ease

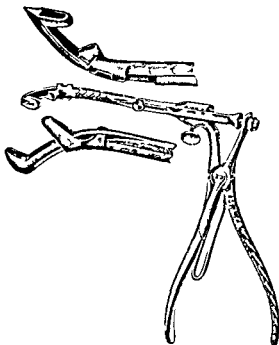


FIG. 136 — Wagner's antrum punches

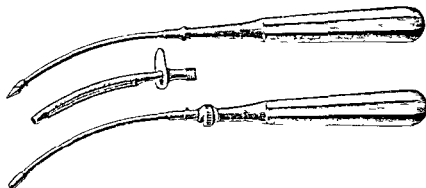


FIG. 137 — Wells' trocar cannula rasp for removing the naso-antral wall

and precision. The instruments which have given the best satisfaction are rasps such as Good's, or punches or biting forceps such as Wagner's or Wells' trocar and cannula rasp.

**Technic.**—Induce local anesthesia of the inferior turbinate and of the inferior and middle meati.

Puncture the naso-antral wall beneath the inferior turbinate about its middle third by means of a trocar or rasp. Then use forward and backward biting punches of the Wagner type to enlarge the opening large enough to overcome the tendency to close. Care should be taken to avoid weakening the attachment of the inferior turbinate. The window should not be carried anteriorly so far that it injures the orifice

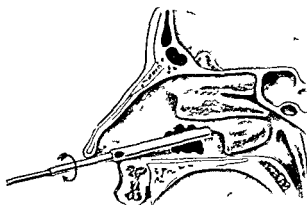


FIG. 138 —The removal of the naso-antral wall with a trephine

of the nasolacrimal duct where it opens under the anterior end of the inferior turbinate. The branches of the sphenopalatine artery in the posterior portion of the inferior meatus should be avoided also.

The first dressing if any is used consists of gauze loosely packed in the maxillary sinus. It should be removed in twenty-four hours. If bleeding is not profuse the packing may be omitted.

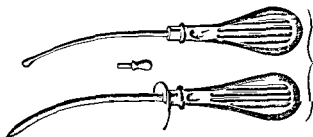


FIG. 139 —Krause's antrum trocar with obturator

In the after-treatment gauze dressings should not be used. The cavity should be left open for drainage and ventilation. Every time the patient blows his nose he blows through the antrum. The antrum should be watched and if exuberant granulations form they should be reduced by the application of chromic acid crystals or other caustic.

## EXTERNAL SURGERY OF THE MAXILLARY SINUS

Internal operations on the maxillary sinus will be described as follows (1) Alveolar (2) Kuster (3) Caldwell Luc (4) Denker (5) Canfield (6) Canfield Billenger

**Removal of the Lining Mucosa** — The question of completely removing the diseased mucous membrane from the maxillary sinus is still a debatable one

Knowlton and McGregor<sup>1</sup> after experimentally removing the lining of the antrum of a dog found epithelial regeneration apparently complete at the end of three months and the subepithelial glands regenerated at five months

Cortes and Lerner<sup>2</sup> after removing the mucosa from the frontal sinuses of a dog found a completely regenerated membrane containing goblet cells and glands after two months and ten days. The mucosa was again removed and seven months later the sinus was lined by a new membrane which appeared normal except for signs of inflammation

Hilding<sup>3</sup> after excising strips of mucosa from the frontal sinuses of dogs reported the formation of ridged scars. He later<sup>4</sup> reported partial or complete obliteration of the sinuses by scar tissue after complete removal of the mucosa. In some cases mucin filled cysts were observed in the scar tissue

Semenov and Kistner<sup>5</sup> found the reformed mucosa not entirely normal

Boling<sup>6</sup> in a study of the regeneration of the nasal mucosa of the lamb found normal ciliated epithelium regenerated by migration with stratification and redifferentiation of the cells; however the tunica propria lacked normal vascularity and the glands did not reach their normal degree of differentiation or number within two months

The evidence presented by the majority of investigations seems to be that the mucosa of the sinuses regenerates in most instances. The regenerated epithelium originates from the nasal mucosa or from the islands within the sinus

**The Alveolar or Cooper Operation** — The alveolar operation was for a long time a popular procedure but is in disuse at the present time due to the danger of establishing a chronic fistula between the antrum and the oral cavity

A devitalized second bicuspid or the first or second molar with an apical abscess is removed and the opening thus made enlarged and its walls rendered smooth. Daily irrigations with warm saline solution followed by penicillin instillations is used for a week or so or until the discharge ceases. If the discharge persists after ten to fourteen days intranasal drainage should be instituted and the alveolar opening permitted to close

<sup>1</sup> Arch Otolaryngol 8 637 (December) 1928

<sup>2</sup> Arch Otolaryngol 12 649 (November) 1930

<sup>3</sup> Arch Otolaryngol 17 321 (March) 1933

<sup>4</sup> Arch Otolaryngol 17 760 (June) 1933

<sup>5</sup> Proc Soc Exp Biol and Med 27 322 (January) 1930

<sup>6</sup> Arch Otolaryngol 22 689 (December) 1935

**The Kuster Operation** — The operation consists of the removal of the anterior wall of the antrum as shown in the Caldwell Luc operation. The opening is usually limited to the area of thin bone of the canine fossa and should be large enough to admit the introduction of the index finger.

With the head mirror light is reflected into the cavity and its walls examined. The portion of the cavity which cannot be inspected should be thoroughly explored with a curved probe.

The preliminary step of the operation consists in the elevation of the upper lip and an incision at the labio-gingival junction (Fig 140). The incision is carried through the periosteum and should be from 1 to 1½ inches in length. The periosteum is then dissected upward over the canine fossa and the upper lip pulled toward the eye with a retractor after which the anterior wall should be removed with a chisel and rongeur bone forceps. The cavity should then be explored

FIG 140 The labio-gingival incision in the Kuster and Caldwell Luc operations

with a probe and the detached mucous membrane and necrotic bone removed with the curette. If the antrum is divided by septa they should be broken down to convert it into one large cavity.

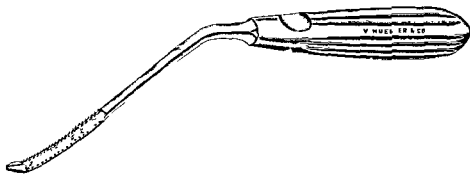


FIG 141 — Thompson's antrum trocar and rap

Having thoroughly removed the infected mucous membrane and tissue a gauze wick is inserted into the canine opening to prevent closure of

the wound. If there is marked suppuration the cavity should be irrigated daily and the wick of gauze introduced to promote drainage. When complete healing has taken place the dressings are discontinued and the labio-gingival opening allowed to close.

**The Caldwell Luc Operation**—This procedure is usually preferable to the Kuster operation. By it the antrum is exposed to direct ocular inspection as in the Kuster operation but in addition a large opening is made in the naso-intral wall.

**Anesthesia**—Local or general anesthesia may be used.

**Technic**—Before beginning the operation the operator should make certain that sufficient room for a naso-intral window exists under the inferior turbinate. If not the turbinate should be fractured medially or in some instances a portion of the turbinate will have to be resected. Any obstruction to drainage through the natural ostium should be removed.

The incision (Fig. 140) is made in the canine fossa at the labio-gingival junction in a horizontal or vertical direction and the periosteum elevated over the canine fossa. Care should be exercised to avoid the vessels and nerves issuing at the infra-orbital foramen. Using suitable chisels, biting forceps and rongeurs an opening large enough to admit the index finger is made through the canine fossa into the antrum. Under direct visual inspection of the interior any diseased portions, tumors, etc. can be seen and removed. All diseased portions of the mucous membrane are removed by inserting variously shaped curettes through the canine opening and gently tearing off the membrane. In using the curette on the roof of the antrum it must be kept in mind that the supra-orbital vessels and nerves are covered only by a thin layer of bone; indeed they may lie bare in the sinus roof due either to disease or to a dehiscence in the infra-orbital canal.

To make the naso-intral window under the inferior turbinate some operators find it easier to make a puncture with a trocar from the nasal side into the antrum and others in the opposite direction. In either case the opening is enlarged by rasps and various direction cutting forceps until the window is at least large enough to admit the thumb and preferably larger. Care should be taken to avoid injuring the inferior end of the nasolacrimal duct anteriorly or the descending branches of the sphenopalatine artery posteriorly. An opening made in the middle meatus instead of the inferior in some surgeons' hands gives good results.

The sinus is lightly packed with gauze saturated with an antiseptic and lubricating ointment. In former years 10 per cent ichthyol, 10 per cent mild silver protein, balsam of Peru have all been beneficial and are still useful. With the development of the sulfonamides and penicillin and in an antrum in which frank pus or infected granulations are found an ointment containing one of the latter is preferable. The packing is brought out through the nose and removed on the first or second day. The last step consists in the closure of the incision in the canine fossa.

The postoperative care consists of a preliminary irrigation of the sinus through the naso-intral window with a saline solution to remove the

clots and gross infection. This is followed by a daily retention wash of penicillin solution (100 units per cc). In more severe infections of the sinus the irrigations with penicillin can be carried out two or three times daily.

In the Caldwell Luc operation some injury to the middle and superior alveolar arteries and nerves is inevitable but this can be kept to a minimum by care in making the canine fossa window. Damage may be done to the roots of the teeth likewise if the canine fossa opening is carried too low.

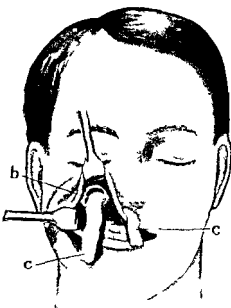


FIG. 142—Applying the dressing after the Caldwell Luc operation. *a* the anterior canine wall removed; *c* the gauze wick in the antrum and extending through the naso-antral opening into the nasal chamber.

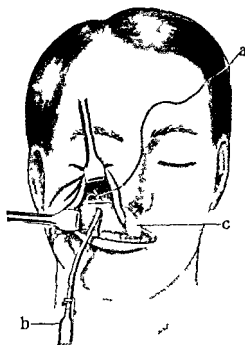


FIG. 143—Closing the labio-gingival incision in the Caldwell Luc operation. *a* the suture; *b* the Re-erdin needle.

It has been claimed that it is unnecessary to do either the Kuster or the Caldwell Luc operation the simple opening through the naso-antral wall being quite sufficient. That the naso-antral opening is sufficient in most of the cases is true. In other cases in which a pronounced degeneration of the mucous membrane and caries of the bony walls of the antrum are present it is necessary to explore the antrum by ocular inspection and curettage a procedure which cannot be successfully done through the nose. The Caldwell Luc operation should therefore be elected in those cases in which there is pronounced suppuration with granulation tissue caries of the bony walls or if tumors are suspected.

**The Denker Operation**—*Indications*—This operation is indicated in those cases where it is necessary to expose the anterior nasal angle

or the naso-antral wall of the maxillary sinus to careful visual inspection and treatment. In the Caldwell Luc operation this anterior angle can not be seen well.

**Technic** — A general anesthetic or block anesthesia should be given. The patient should be placed in Rose's position with the head hanging over the end of the table if a general anesthesia is used. In the latter event postnasal tampons should be introduced to keep the blood from the throat and trachea.

The labio-gingival incision should be made as in the Caldwell Luc operation but should extend to the median line. Some operators prefer a vertical incision.

Elevate the soft tissues and periosteum over the canine fossa.

Remove the anterior wall (canine fossa) of the maxillary sinus as in the Kuster and Caldwell Luc operations and then remove the bridge of bone between the canine fossa and the lower portion of the pyriform opening of the nose as shown in Figure 144. By thus extending the bony wound the anterior angle of the sinus is exposed to operative interference.

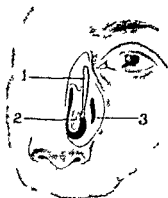


FIG. 144 — Showing the relation of the lacrimal duct to the inferior turbinate. 1 the lacrimal duct 2 the inferior turbinate 3 the maxillary sinus (after Bardeleben)

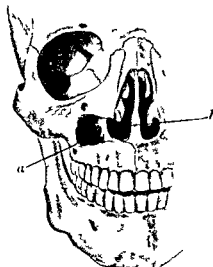


FIG. 145 The Denker antrum operation. a the area of bone removed in the Kuster and the Caldwell Luc operations. In the Denker operation additional bone is removed from b to the pyriform aperture.

Through the opening thus made remove the diseased membrane and granulation tissue.

Elevate the mucoperiosteum of the inferior meatus of the nose and of the inferior turbinate with a small flat elevator so curved as to adapt it to the anatomic configuration of the part.

Incise the mucoperiosteum thus elevated so as to convert it into a rectangular flap to be turned outward on the floor of the sinus. The flap is held in position for forty-eight hours with a gauze dressing.



The opening in the naso-antral wall should be quite as large as in the Caldwell Luc operation or otherwise it may close and drainage from the sinus precluded. The inferior turbinate should be fractured inwardly if it blocks the opening, or in rare instances it may be necessary to resect a portion of it.

The after-treatment as in the Caldwell Luc operation consists of watching the antrum and reducing exuberant granulations with caustics as soon as they appear.

The objection to the Denker operation is that in many cases the infra-orbital and anterior dental nerves may be injured thereby devitalizing the teeth or producing areas of anesthesia.



FIG. 146—Canfield Ballenger operation. *a* the margin of the pyriform aperture the point of incision for the Canfield Ballenger antrum operation.

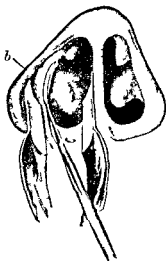


FIG. 147—Canfield Ballenger operation. *b* the incision.

**Canfield Ballenger Antrum Operation**—The following technic is a slight modification of the Canfield operation as originally described by him but differs inasmuch as the inferior turbinate is preserved.

Entrance into the antrum is gained by removing the inferior lateral angle of the bony pyriform aperture as in the Denker operation but unlike the latter is done through the nose.

**Technic—Anesthesia**—(*a*) Induce anesthesia of the nasal mucous membrane by the local application of cocaine (*b*) induce anesthesia of the vestibular skin of the nares by the injection of 1 or 2 per cent procaine. This solution should also be injected beneath the periosteum of the canine fossa *via* the vestibule of the nose.

**Incision**—Distend the wing of the nose with a nasal speculum as shown in Figure 146 *a* to bring the anterior angle of the naso-antral wall into prominence. Then with a small scalpel make an incision the whole length of the exposed portion of the naso-antral angle (margin of the pyriform aperture) (Fig. 147 *b*). Then elevate the membrane including the periosteum over the canine fossa.

*Opening the Naso-antral Angle* — The antrum should be opened via the naso-antral angle (ungula pyriformis) with rongeur bone forceps as shown in Figure 148 *c* or with a gouge and mallet. In some subjects the bone at this angle is dense requiring considerable force to bite through it while in others it is extremely thin and easily removed. While the incision extends higher than the attachment of the inferior turbinate (to allow retraction) the bone at the angles should be removed only below the line of attachment of the inferior turbinate. In removing the bone constituting the canine fossa it is usually necessary to remove only enough to admit of the introduction of the Wagner antrum forceps as shown in Figure 149 *d*. If however after making the opening through the naso-antral angle it is determined that the whole of the mucous membrane is not accessible to the curette as much of the canine wall may be removed as will fully expose it.

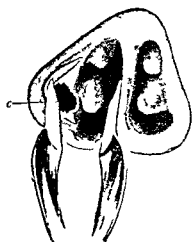


FIG. 148 — Canfield Ballenger operation on the naso-antral angle removed thereby exposing the cavity of the antrum

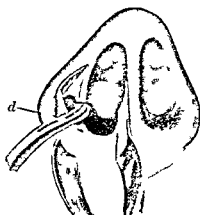


FIG. 149 — Canfield Ballenger operation of the naso-antral angle being secured with the Wagner forceps

*Removal of the Naso-antral Wall* — The biting jaws of the Wagner forceps are placed astride the naso-antral wall and the wall bitten away from the attachment of the inferior turbinate down to the floor (Fig. 150).

If the mucous membrane of the antrum is edematous and has undergone polypoid or granulation degeneration it is necessary to remove it.

If bleeding is persistent gauze packing should be introduced. This should be removed within twenty-four hours and renewed if necessary.

### OROMAXILLARY FISTULA

*Etiology* — An oromaxillary fistula is usually due to the removal of a tooth which is in close relationship to the floor of the maxillary sinus. The root of the tooth may extend into the sinus or close enough to the floor of the sinus to result in a fracture or breaking of the sinus floor when the tooth is extracted. If the root of the tooth is not infected the fractured floor of the sinus may heal without an infection developing.

in the maxillary sinus. In most instances however the tooth root is infected and an odoriferous sinusitis develops which may result in a persistent oromaxillary fistula through the tooth socket. The fistula is more apt to develop if long continued irrigations of the sinus is carried out through the tooth socket.

Other and less common causes for an oromaxillary fistula are infections, cysts and neoplasms of the maxilla which may destroy the intervening bone, resulting in a fistula between the maxillary sinus and the oral cavity. A fistula may follow traumatic injuries of the maxilla in which the lining mucous membrane of the sinus is torn. Osteomyelitis and necrosis of the maxilla especially in infants may result in an oromaxillary fistula as well as fistulae in other regions.

**Symptoms**—The symptoms of an oromaxillary fistula if of recent origin is an escape of blood into the nostril or an escape of air from the tooth socket into the oral cavity. If blood is oozing from the tooth socket it is usually frothy. Liquids taken into the mouth may escape through the nostril.

If an infection develops in the sinus as it usually does within twenty-four to forty-eight hours following the extraction of the tooth symptoms and signs of an acute maxillary sinusitis supervene. The sudden onset of an odoriferous nasal discharge is characteristic. If the fistula is chronic the symptoms and signs of the sinusitis are those of a chronic infection except the odor is persistent and a discharge of pus and air is noticed coming from the fistulous tooth socket.

**Treatment**—Any maxillary sinus infection must be eradicated first and this alone may permit the fistula to close if of recent origin. Any foreign object in the sinus such as a tooth must be removed. Stimulation of scar tissue around a small fistula may effect a closure. In larger openings or in the event other methods have failed a sliding mucous membrane flap after excision of the scar tissue will be necessary. In some instances it is necessary to close the oral opening before the sinusitis will heal. In any event adequate drainage of the sinus through the nose must be maintained.

**Sliding Flap Operation**—A number of somewhat similar techniques based on the sliding flap principle have been devised to close an oromaxillary fistula. The flaps are taken from the palate or from the buccal surface of the alveolar ridge or in some instances flaps from both regions are used. Dunning<sup>1</sup> uses a pedicle flap from the palate and sutures it to a labial mucosal flap. Welty<sup>2</sup> makes a median incision in the palate then elevates the palatal mucosa between the incision and the fistula and after reducing the alveolar process approximates the palatal flap to a small labial flap. Ashley<sup>3</sup> uses a mucoperiosteal flap from the palate denudes the anterior portion of mucous membrane then plugs the fistular opening with the denuded end of the flap. Hill<sup>4</sup> turns back a flap from the gingival mucosa adjacent to the fistula to form a lining for the opening then covers the raw surface of the turned back flap.

Laryngoscope 35 766 (October) 1925

<sup>1</sup> Trans Sect Laryngol Otol and Rhinol Am Med Assn 1900

<sup>2</sup> Trans Am Laryngol Rhinol and Otol Soc 45 440 1939

<sup>4</sup> Arch Otolaryngol 40 167 (September) 1944

with a sliding flap from the buccal mucosa. For large defects Hersh<sup>1</sup> uses a technic similar to Hill's but rotates a large buccal flap 180 degrees on its longitudinal axis so that its epithelial surface is presented to the interior of the antrum and the raw surface toward the oral cavity. The flap is sewed into place with fine silk. A flap from the palate is then sutured over the raw surface of the buccal flap. At the end of the third week the bridge of tissue created by the rotated buccal flap is severed near the outer edge of the alveolar ridge. The pedicle is then rotated back to its original position and sutured in position.

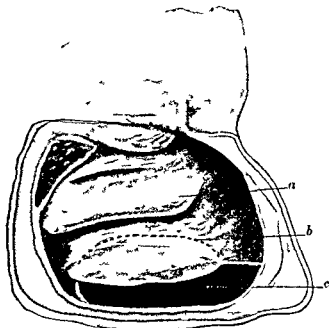


FIG. 150.—Interior view of the Canfield-Ballenger antrum operation: *a*, middle turbinate; *b*, line of attachment of the inferior turbinate which is left intact; *c*, the naso-antral wall removed extending from the floor of the nose to the attachment of the inferior turbinate and from the anterior to the posterior limits of the antrum.

Proctor<sup>2</sup> first cures the fistulous tract, then inserts a shaped plug of preserved rib cartilage into the fistula so that a tight fit is obtained. The portion of the cartilage external to the bony opening is cut level to the surrounding bone. A mucous membrane flap to cover the exposed cartilage was not found essential to complete healing of the fistula. The associated infection in the maxillary sinus should have proper attention.

### INTRANASAL SURGERY OF THE ETHMOID SINUS

**Mosher's Intranasal Ethmoidectomy**—This operation has proved an excellent and satisfactory procedure and appears to be the simplest and safest operation devised for its purpose. It is adaptable to exenteration of the anterior ethmoid cells alone with probing and enlargement of the frontonasal duct or to the removal of the anterior and posterior

<sup>1</sup> Arch Otolaryngol 43:141 (February) 1946.

<sup>2</sup> Laryngoscope 56:46 (February) 1946.

ethmoid cells. It will reduce the necessity of performing the external operation in many instances even though the latter is the easier, safer and more thorough method.

**Technic** — A local or general anesthetic can be used. Introduce an ethmoid curette into the nasal chamber until the cutting edge of the instrument facing the orbit is above the anterior attachment of the middle turbinate. This area covers the frontonasal canal and the anterior ethmoid cells draining into it. The bone at this point is usually very thin and easily broken down. Having located the instrument make

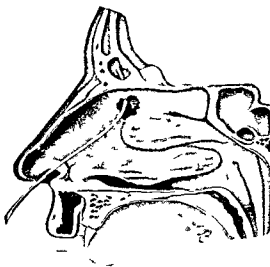


FIG. 151 — The lateral nasal wall just above the anterior attachment of the middle turbinate has been broken down and a probe has been inserted through the fronto-nasal duct into the frontal sinus.



FIG. 152 — The ethmoid cells are removed by introducing a curette through the opening made in the lateral nasal wall and sweeping it downward and backward between the orbital and turbinal plates of bone.

gentle but firm pressure toward the orbit until the curette breaks into the labyrinth. Then turn the curette so that it faces anteriorly and withdraw it in a sweeping movement anteriorly and downward. A few such movements with the curette will give the result shown in Figure 131. By continuing the curettage in a forward and downward direction the frontonasal duct is enlarged and free drainage of this sinus established. The frontal sinus may now be entered with a blunt pointed frontal sinus probe. Indeed in most instances a suitably bent cotton wound applicator may be easily introduced.

Further curettage in this area would complete the anterior ethmoidectomy but the removal of all the anterior ethmoid cells is difficult due to their variation and wide distribution. Occasionally one or more of the cells extend over the orbital roof posterior to the frontal sinus. In other cases a cell encroaches upon the floor of the frontal sinus and forms the so-called bulla frontalis. The dense nasal bone and nasal process of the frontal bone which form the anterior wall of the ethmoid labyrinth often shield some of the most anterior of the cells making access difficult by the intranasal route.

The next step in the operation is the removal of the posterior ethmoid cells. This is done with the same curette introduced through the opening already made as shown in Figure 132. The curette is introduced beneath the cranial plate or roof of the ethmoid labyrinth and then brought downward between the lateral (orbital) and medial (nasal) walls or plates. This procedure is repeated several times until the anterior wall of the sphenoid sinus is reached. In using the curette the operator must always bear in mind the position of the cribriform plate. The cribriform plate is located at the roof of the nasal cavity—that is medial to the lateral nasal wall. It may be superior or inferior to the level of the roof of the ethmoid labyrinth. It may be exposed by disease. The curette should always be kept lateral to the lateral nasal wall to avoid injuring the cribriform plate. If the anterior ethmoid vessels are encountered they will indicate the level of the cribriform plate. Anterior to the frontonasal canal there is less danger because the cribriform plate does not extend that far forward.

The entire posterior and anterior ethmoid regions are again examined by ocular and probe inspection and all available cells are removed. The posterior ethmoid cells may remove all directions so that except under favorable conditions a complete removal is not possible by the intranasal route.

The last step in the operation is the removal of the middle and superior turbinates. This should be done with great care using a suitable biting instrument where possible to sever the superior attachment of the turbinate bones. When the superior margin of the superior turbinate has been severed the remaining inferior portion can be gently rocked and teased free without fear of fracturing the cribriform plate (Fig. 153).

At the conclusion of the operation all loose particles of bone and mucous membrane should be removed. If frank pus or infected granulation tissue containing susceptible organisms are encountered penicillin

powder or one of the sulfonamide powders should be insufflated and repeated daily until the infection has been overcome. Penicillin solutions containing 500 to 1000 units per cc. can be used to irrigate the



FIG. 153—The superior and middle turbinates are grasped with forceps and with the traction combined with a rocking motion detached and removed from the nose.

ethmoid area postoperatively if desired. Crusting and drying of the mucous membrane is counteracted by daily applications to the operated area of pledgets of cotton saturated with 10 per cent ichthol or 10 per cent mild silver protein and left in place for about fifteen minutes.

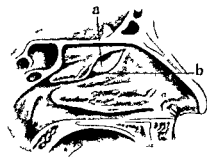


FIG. 154—Showing a large bulla ethmoidalis (a) encroaching upon the hiatus semilunaris (b), the hiatus semilunaris. The anterior half of the middle turbinate has been removed.

Hemorrhage is an occasional complication attending the ethmoid operation. A postoperative pack need not be used unless bleeding does ensue and should be left in place for twenty-four hours only. The ethmoid labyrinth is supplied chiefly by the anterior and posterior ethmoid arteries and hemorrhage when it occurs is from them. Snug packing with petrolatum-treated gauze in the cavity is usually sufficient.

Ordinarily healing is well progressed at the end of ten days and if the eventration has been complete the space in the ethmoid region should be free and roomy and the frontonasal duct have free access to the nasal cavity.

### INTRANASAL SURGERY OF THE SPHENOID

Surgical procedures upon the sphenoid consist in making an opening into or removing the anterior sphenoid wall.

The preliminary operative procedure for reaching the sphenoid sinus

usually consists of the partial or complete removal of the middle turbinate thus exposing the sphenoid ostium to view.

The Hayek, Fletcher or other sphenoid forceps may be used. One of Hayek's forceps cuts upward and the other downward. Fletcher's forceps cuts in all directions as its biting end is a circular disk. If the ostium is small it should first be enlarged with a curette. The upward cutting forceps should then be introduced and the upper portion of the wall removed. By turning the forceps to either side the lateral portion of the wall may be removed. Next introduce the downward cutting forceps (Fig. 100) and remove the lower portion of the wall. The wall near the floor of the sinus is quite thick but is readily removed with Hayek's forceps. When the wall is entirely removed the opening is often  $\frac{1}{4}$  x  $\frac{3}{4}$  inch in area and the interior of the sinus may be inspected by reflected illumination or by a nasopharyngoscope. When the mucous membrane is normal it is pink and by contrast with the nasal mucous membrane appears almost white. Under probe pressure it is thin, firm and slightly resilient. When infected it is more red, edematous and thickened. In some cases the sinus is filled with granulation tissue or polyp.



FIG. 100.—Removing the anterior wall of the sphenoid sinus with the Hayek forceps. The distal blade of the forceps is introduced through the sphenoid ostium and the bony wall removed by successive bites.

The after treatment consists of saline irrigations and the topical application of a 10 per cent aqueous solution of ichthylol. As there is a marked tendency for the mucous membrane to reform over the opening in the sinus it may be necessary to remove it from time to time to maintain ventilation and drainage. This is easily accomplished as the middle turbinate has been previously removed and the tissue to be removed is membranous. The after treatment may extend over many weeks.

Hemorrhage following an operation on the sphenoid may come from the sphenopalatine artery and the posterior lateral nasal artery. The sphenopalatine artery leaves the sphenopalatine foramen to run along the lower portion of the anterior wall of the sphenoid to the septum. The posterior lateral nasal arteries leave the sphenopalatine to break up into branches supplying the turbinates and posterior ethmoid cells. These vessels are injured when working near the posterior tip of the middle turbinate.



## CHAPTER XVI

### THE COMPLICATIONS OF SINUS DISEASE

#### RETROBULBAR NEURITIS

. **Etiology.**—Retrobular neuritis may be due to disease of the nasal accessory sinuses, more particularly the ethmoid and sphenoid sinuses

In the etiology of retrobulbar neuritis, multiple sclerosis probably is the most important cause. Gifford believes cases due to purulent sinusitis comprise about 3.5 per cent. Of the 15 or 20 per cent for which no other cause can be found a latent or hyperplastic sinusitis should be considered as there is pathologic evidence that such forms of sinusitis may cause retrobulbar neuritis by direct extension.

Benedict<sup>1</sup> in an analysis of the etiology of 225 cases of retrobulbar neuritis at the Mayo Clinic found multiple sclerosis accounted for the great majority (155 cases). A brain tumor may be a factor in rare instances. In addition abscesses at the apices of teeth, absorption of toxic material from the intestines, infected tonsils, vascular disease, syphilis, tuberculosis, inhalation or ingestion of poisons such as lead, arsenic, acetone, alcohol, tobacco, etc.; the acute infectious diseases, including erysipelas, mumps, influenza, tonsillitis, measles, pneumonia and malaria, may be the factor producing the pathology.

**Pathology.**—The optic nerve may be involved from a sinusitis either by pressure, extension of the inflammation, vascular congestion, or by a thickening process produced by an osteitis or periostitis with pressure on the optic nerve. Neivert found the optic nerve in 75 per cent of his specimens caused the roof of the sphenoid sinus to stand out in relief, with pockets at either side. There were 12 specimens (out of 220 examined) with dehiscences so located as to expose the nerve.

In some instances a direct extension of an acute inflammatory infection in the posterior sinuses may extend rapidly by continuity of tissue to the sheath or to the optic nerve itself.

White believes the size and shape of the optic canals have some relation to the incidence and severity of retrobulbar optic atrophy. A canal of 4 mm. or less in a patient with severe optic atrophy not due to other causes suggests immediate ventilation of the sinuses. A canal of 4.5 mm. gives more time for study, while one that is 5 mm. or over will recover from a very acute attack without operation.

**Treatment.**—In retrobulbar neuritis there is a tendency toward spontaneous cure. Before resorting to a sinus operation in these cases, every effort should be made to eliminate other causes, especially multiple sclerosis and all foci of infections and other toxic agents. However, if a patient has an increasing retrobulbar neuritis for which no other cause

<sup>1</sup> Arch. f. Ophth. 9, 893, (June) 1933

can be discovered and for which there is reason to suspect sinus disease it is considered justifiable to explore the ethmoid and sphenoid sinuses

# ORBITAL CELLULITIS AND ABSCESS

**Orbital Cellulitis** — The most frequent cause of orbital cellulitis in children is sinusitis

The anatomic conditions in children which contribute to the development of orbital complications are congenital dehiscences along the ethmo maxillary suture in the lamina papyracea or in the orbital wall of the maxilla. There is a more profuse development of the lymphatic and vascular system than in an adult as a result infection from the sinuses may enter the orbit along the veins as a thrombophlebitis without any bony perforation

Pus in all cases does not perforate into the orbit it may be guided anteriorly beneath the periorbita until it points as a rule through the

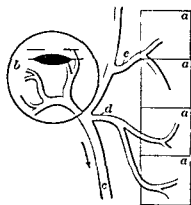


FIG 15c — Schema showing the venous connections of the ethmoid cells with the eyeball. *a a a* anterior and posterior ethmoid cells. *b* eyeball. *c* superior ophthalmic vein. *d* posterior ethmoidal vein. *e* anterior ethmoidal vein.

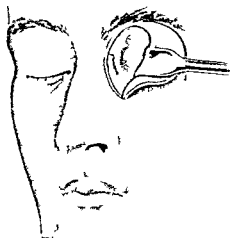


FIG 15 — Exposure of the anterior ethmoid cells through the inner wall of the orbit. The method of procedure is adapted to the cases complicated by orbital cellulitis.

inner portion of the upper eyelid where it sometimes ruptures spontaneously (Fig 15d). If the maxillary sinus produces the orbital cellulitis, pointing usually occurs through the inner portion of the lower lid.

Other causes of orbital cellulitis are traumatic teeth, osteomyelitis, erysipelas, meningitis, brain abscess, the exanthems, metastasis, and actinomycosis.

The first symptom of orbital cellulitis is edema of the eyelids which increases very rapidly. Chemosis is usually present. Proptosis may take place and this often affords a valuable clue as to the location of the pus. Limited extra ocular movements occur as the cellulitis or

abscess produces pressure within the orbit. Examination of the fundus usually discloses dilated retinal veins. Indentation of the globe simulating detached retina, choked disk, optic atrophy, optic neuritis, retinal hemorrhages or retrobulbar neuritis may be present in rare instances. The value of the roentgen ray in the diagnosis of the orbital condition is limited. *Streptococcus hemolyticus* is found in many cases.

**Orbital Abscess With Exophthalmos** — **Etiology** — Orbital abscess with exophthalmos as a complication of nasal sinusitis is rare. When present it is almost invariably unilateral. The most frequent cause of the exophthalmos is orbital cellulitis or abscess secondary to a frontal ethmoid or maxillary infection involving the former in adults and the two latter sinuses in children.

**Pathology** — The mode of infection of the orbit from nasal sinus disease may be from necrosis in the common bony wall of the nasal sinus and the orbit or from a thrombophlebitis of the veins of the diseased nasal sinus. Other causes such as dehiscence and trauma following operations on the nasal sinuses are rare.

**Symptoms** — The clinical picture with external signs is definite. After a suppurative rhinitis, an edema of the lid and adjoining region, a livid color of the skin of the lids, a loosening of the tarsus and a protrusion

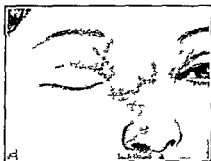


FIG. 158.—Pterygoid abscess secondary to an ethmoiditis. The abscess is pointing at the inner canthus of the eye.



FIG. 159.—Pterygoid abscess secondary to an ethmoiditis after incision and drainage.

of the bulb due to periorbital involvement develop. The conjunctivae are not injected or swollen. There is always malaise, high fever and loss of appetite, this syndrome pointing to a severe involvement. Especially frequent in children are the large vesicles which are in marked contrast to the bluish discoloration of the eyelids.

In a few instances the abscess may discharge spontaneously either through the nostril or externally.

**Diagnosis** — The differential diagnosis must be made from inflammation or orbital phlegmon caused by trauma and by infection about the head other than nasal such as osteomyelitis of the superior maxilla and from that due to metastasis from remote septic conditions, orbital neoplasm, syphilitic gumma and periostitis, vascular tumors, orbital hemorrhage, cavernous sinus thrombosis, exophthalmic goiter, mucocele and pyocoele.

The non-inflammatory or neoplastic type of exophthalmos due to a mucocele, pyocele or tumor of the frontal or ethmoid sinuses is characterized by a gradual onset without any signs of phlegmon, a chronic course with gradual development of exophthalmos and dislocation of the eyeball unaccompanied by pain or sensitiveness.

**Complications**—The pus may burrow along the periorbita to the sheath of the optic nerve and result in an intracranial complication. An extension of a thrombophlebitis of the orbital veins into the cavernous sinus will result in a cavernous sinus thrombosis.

**Treatment**—The patient should be placed immediately on full doses of one of the sulfonamides and or penicillin. Hot compresses should be applied to the orbit and tampons of 1 to 3 per cent ephedrine placed in the middle meatus beneath the middle turbinate for five to ten minutes to promote drainage from the sinus. In most instances this treatment is sufficient. If a collection of pus is present incision and drainage is indicated. A 1 inch type of incision below the eyebrow is usually done. The periosteum is elevated from the superior orbital wall until pus is encountered. If the periorbita is intact care should be taken to avoid puncturing or tearing it while elevating otherwise an infection of the orbital contents may occur.

### OSTEOMYELITIS OF THE FRONTAL BONE AND SKULL

Osteomyelitis of the frontal bone and skull is reported in the literature is somewhat rare. Furstenberg found but 73 cases up to 1931. However it is probable that only a small proportion of the total number of cases have been reported.

**Etiology**—The organism recovered in the great majority of the reported cases is the *Staphylococcus aureus*. The *Streptococcus pneumoniae* and anaerobic streptococcus are found in a few instances.

The etiology depends to a certain extent upon the virulence of the organism and the resistance or immunity of the patient to the particular organism present.

In children the origin of osteomyelitis of the frontal bone is almost always hematogenous. In adults the disease is more likely to result from trauma or by spread from an adjacent infection.

The majority of patients are under thirty years of age. It is more common in females than in males. Many cases follow swimming. (*Chronic infection of the sinuses may predispose to osteomyelitis especially following an acute exacerbation.*) Trauma in the region of the frontal sinus or operative trauma following surgical procedures on the frontal bone frequently precedes the advent of osteomyelitis. It has been reported as following operations on the maxillary sinus. Operating in an infected field, traumatizing the bone, closing the external wound too tightly, rasping the bone and operating during an acute exacerbation of a chronic infection have been mentioned as possible factors.

**Pathology**—The infection may be transmitted to the vault by continuity of tissue or as is more common by hematogenous metastasis.

In the latter event the inflammatory disease is carried by a thrombophlebitis of the venous system into the bone of the calvarium through

the anastomosing diploetic veins through the frontal sinus or on the under surface of the cranium or the frontal sinus. Isolated foci of osteomyelitis in remote bones of the skull may be accounted for by this later method. Furstenberg<sup>1</sup> found the inner plate the first to be affected while the external plate may remain uninvolved although the reverse may be true as well. The intracranial extension of the infection is usually by way of the frontal and anterior temporal diploetic veins into the superior sagittal sinus and from there to the cerebral hemisphere of the same or opposite side by way of the connecting veins.

Infection by continuity of tissue is probably the less common form of extension. Direct invasion of the canaliculi and medullary spaces seems to occur especially in the postoperative cases. The cranial sutures in many instances exercise an inhibiting influence upon the spread of the infection. In rare instances spread of the infection along the perineural sheaths of the olfactory nerve may occur (Courville and Rosenwald<sup>2</sup>).

In the earliest stage of osteomyelitis of the cranial bones the diploes show some congestion. Somewhat later marked hyperemia with small drops of pus may be seen. Its consistency is softened and the diploetic spaces are filled with granulation tissue bathed in pus. Thrombosed vessels may or may not be found. The bone itself becomes discolored with blood and pus oozing to the surface through the vascular channels or fistular openings. Small sequestra occur in the diploetic spaces. The external and internal tables may be broken down with widespread destruction.

Microscopic examination in the early stage shows edema of the myeloid tissue with a vascular congestion and an infiltration of lymphocytes and polymorphonuclear leukocytes. Some of the vessels may show a septic thrombosis. Later necrotic areas are found the result of an obstruction to their blood supply. The myeloid tissue is replaced by granulation tissue and pus. Osteoclasts may surround the sequestra and be seen along the surface of the bone. Evidence of osseous repair and areas of destruction are present at the same time.

The infection may extend along the dura, the periosteum and the soft tissues of the scalp at about the same rate. If the infection breaks through the inner table an extradural abscess is formed. Furstenberg believes this abscess is responsible for the further extension of the infection by cutting off the blood supply of the cranial bones.

**Symptoms**—The clinical course may be acute or chronic. The symptoms depend on the course, the sinus involved and the extent of involvement.

In the acute fulminating type fever, headache and edema of the upper eyelid on the affected side are present. The soft doughy swelling (Pott's puffy tumor) or pericranial abscess is pathognomonic of osteomyelitis of the underlying bone. This type frequently follows swimming. It shows a tendency to early spread to the intracranial structures. Death

<sup>1</sup> Ann Otol Rhinol and Laryngol 45:726 (September) 1936.

<sup>2</sup> Arch Otolaryngol 27:692 (June) 1938.

may occur from a few days to a week or two from severe toxemia or meningitis. As a rule however osteomyelitis is a slow disease even in the acute cases. Months may elapse before a cure is effected or death occurs. Cyclic exacerbations are characteristic.

The chronic localized form without perforation of the internal table is characterized as a rule by an insidious onset, a low-grade fever, local pain or tenderness, doughy swellings, general malaise and occasionally chills.

In the chronic form of osteomyelitic fistula sequestra and purulent discharge from the bone may be present with cyclic exacerbations.

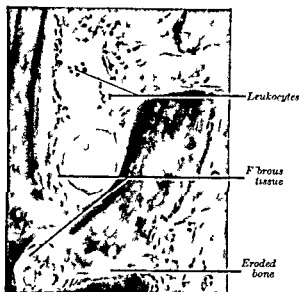


FIG. 160. Osteomyelitis of the frontal bone ( $\times 700$ ). The term osteomyelitis indicates an inflammation of bone and bone marrow but is essentially an inflammation of the soft parts of bone (periosteum plus the contents of the medullary cavity and Haversian canals).

**Diagnosis**—The diagnosis is made from the roentgenogram combined with the signs and symptoms of fluctuating swellings, advancing edema, persistent low grade temperature, leukocytosis and pain and heralded by cyclic exacerbation.

The differential diagnosis should be made from acute sinusitis, syphilis and tuberculosis.

**Prognosis**—The mortality in the groups of reported cases has been high. It has varied from 35 to 79 per cent. It is much higher in the postoperative than in the spontaneous types. The sulfonamides and the antibiotics have decreased the incidence as well as the mortality.

**Treatment**—The conservative treatment consists of draining the abscesses as necessary, of operating when the acute infection is quiescent, limiting the operation to the removal of sequestra and part of the infected bone. The periosteum is not elevated more than necessary.

Supportive treatment consists of the sulfonamides and/or penicillin.

both local and general is indicated blood transfusion, the administration of iron tonics such as 0.6 gm. of reduced iron tablets daily, ultra violet and infrared radiation, vaccines and intravenous coal liver oil and dressings of bacteriophage.

Williams and Heilman<sup>1</sup> report the finding of an aerobic streptococcus in two cases of osteomyelitis of the frontal bone with favorable results from specific therapy with an autogenous antiviral virus.

In the localized type delay in operative intervention is advisable until the infection has become walled off and the diseased bone has separated as a sequestrum. The involved portion and the sequestrum is lifted out through an incision. Many eminent rhinologists advocate the radical removal of all the infected bone plus a wide margin of normal bone as soon as the diagnosis is made in all types. The relative merits of the conservative and radical measures have not been determined exactly at the present time.

In the slowly spreading type with no tendency to complete localization or if the lesion shows a tendency to advance at any time a radical removal should be instituted.

In the radical operation an immediate and entire removal of the diseased bone is done. The whole thickness of the bone is removed down to the dura. The bone resected must extend beyond the obvious limits of the disease. Mosher and Judd<sup>2</sup> believe that the obvious limit of the disease is the upper limit of the edema. They prefer the mid line inverted T incision with the reflection of two triangular skin flaps, however an incision through both eyebrows over the bridge of the nose extending backward and upward into the scalp gives a free exposure with less visible scarring. Less scarring is also obtained by a coronal incision above the scalp hairline. This permits a broad inverted U shaped flap to be reflected downward uncovering the frontal bone. The anterior and posterior walls of the frontal sinus are removed. The wound is left open but packed with vaseline gauze. Covering the operative defects with vitallium metal plates has been quite satisfactory in the short time it has been used.

The operation may have to be done in two or more stages because of the shock and hemorrhage. Blood transfusions may be given before and after operation if indicated.

Regeneration of the bone seems to occur slowly taking from one to five years for complete regeneration. A plastic surgical procedure may be attempted after one or two years when latent organisms in the tissues have disappeared.

**Complications**—The intracranial complications may be extradural abscess meningitis occasionally frontal lobe or cerebral abscess thrombophlebitis of the superior longitudinal sinus and intradural abscess.

### OSTEOMYELITIS OF THE SUPERIOR MAXILLA

Acute osteomyelitis of the superior maxilla in infants is occasionally seen secondary to a probable buccal infection. Involvement of the

<sup>1</sup> Arch Otolaryngol 25 196 (February) 1937

<sup>2</sup> The Laryngoscope 63 153 (March) 1933

dental sac follows with extension of the necrotic process to the walls of the maxillary antrum creating a pus discharge into the nose and mouth. The infection seems to travel by retrograde thrombosis which is the cause of the gangrene and sequestration. Lederer<sup>1</sup> believes the associated acute osteomyelitis is the result of the venous infection—not the cause of it. He bases his opinion upon carefully studied serial sections of a case in which a nasal infection and sinusitis were found to be the primary cause of the osteomyelitis of the maxilla in an infant.

Osteomyelitis of the maxilla in nurslings and infants may occur from the first week up to the ninth month. The greatest incidence is during the first three weeks. The portal of entry and the manner of spreading of the primary infection may vary.

As shown by Lederer a sinusitis may produce a periosteitis and osteitis with a fistulous tract formation which may extend in any one of three ways: (1) To the facial surface with swelling of the soft parts of the cheek, breaking down of Bichat's pad and abscess formation. (2) to the palatine and alveolar process with a fistula into the roof of the mouth. The tooth anlage may be extruded. (3) to the zygomatic process with a necrosis of the zygomatic arch and extension into the pterygoid fossa with abscess formation. Extension along the fascial planes to the mandibular foramen may occur. An ethmoiditis may result in a periosteitis, osteitis and periorbital cellulitis which may extend in one or both of two ways: (1) Thrombophlebitis of the venous channels with extension to the cavernous sinus and the production of a thrombosis. (2) a periorbital abscess may form with an external fistula.

**Symptoms**—The signs and symptoms are those of a sinusitis accompanied by marked swelling and chemosis of the cheek. Exophthalmos with limitation of motion may be present.

The first or septicemic stage may last for about ten days with the formation of fistulae in the infraorbital regions, palate and in rare instances in the nose. This is followed by the second or chronic indolent stage with persistence of the fistulae and with sequestration of dead bone. The second stage may last for several months especially if free drainage has not been instituted at an early period.

The infant may succumb at an early period from septicemia or bronchopneumonia or later from a brain abscess. There is a mortality of about 25 per cent (Asherson).

The disease should be differentiated from orbital abscess, uncomplicated maxillary sinusitis and tear sac infection.

**Treatment**—The supportive treatment should consist of adequate doses of the sulfonamides and or penicillin as indicated. Free surgical drainage through the mouth or at times through the maxillary sinus should be instituted. Drainage through the cheek over the superior maxilla should be avoided if possible.

### OSTEOMYELITIS OF THE SPHENOID

Osteomyelitis of the sphenoid bone is rare. A few cases have been reported in recent years. Many cases are associated with osteomyelitis



of the base of the skull or are secondary to an infection of the petrous portion of the temporal bone. Eagleton<sup>1</sup> attributes the rarity of infection of the base of the sphenoid to the preponderance of red cellular bone-marrow found throughout life in this bone.

The organisms usually recovered are the pneumococcus type III and the hemolytic streptococcus.

Infection when present in the marrow spreads as a phlebitis or a periarthritis and produces an obliterating vasculitis.

**Symptoms** — The early symptoms, if any are present, would be those of an acute or chronic sphenoiditis such as a postnasal discharge or inflammation, deep seated headache behind the eyes or possibly radiating to the temporal or occipital regions.

Like symptoms such as photophobia, scotomas or blepharospasm may be present.

Later as the body of the sphenoid becomes more extensively invaded symptoms of sepsis ensue although the temperature may be low and the toxemia not marked. The most characteristic late symptom is the marked retro-orbital or temporal pain especially severe at night. Bacterial invasion of the blood stream usually occurs.

**Complications** — The complications are meningitis, thrombosis of the cavernous sinus, septicemia, abscess of the brain, encephalitis and intracranial hemorrhage. Erosion of the sphenoid roof is frequent or an extension along thrombosed vessels to neighboring structures may take place. Erosion may occur at times through the posterior surface where the body is in relation to the basilar part of the occipital bone.

## MUCOCELE AND PYOCELE OF THE NASAL ACCESSORY SINUSES

Mucocele of a sinus is the accumulation and retention within it of mucus and mucoid secretion with blocking of the sinus ostium, and with thinning of the bony walls of the sinus with possible distention of one or more of its walls. When pus is present it is known as suppurative mucocele or pyocele. Any one of the paranasal sinuses may be affected although the sphenoid sinus is rarely so. It is usually confined to the frontal sinuses.

**Etiology** — Mucocele of the frontal sinus is usually seen in late middle life but may occur in youth.

The etiology has not been established. Two theories have been advanced. (1) It is due to an enlargement of a retention cyst of one or more mucous glands or cystic degeneration of a polyp. (2) it is the result of a closure of the nasofrontal duct from obstruction, inflammation or trauma.

**Pathology** The wall and mucous membrane lining of a mucocele show changes due to pressure. The mucosa is thin and the columnar epithelium may be flattened with deficient or absent cilia except for occasional isolated areas. The mucous glands are dilated in the early stage but followed by atrophy later. In the late stage the sac wall is

thickened with fibrosis, epithelial hyperplasia and capillary proliferation

The bony walls surrounding the sac show areas of rarefying osteitis and erosion with other regions giving evidence of proliferative changes especially around the margins of the sinus

The mucocele contains a tenacious viscid or gelatinous mucus yellow or greenish brown in color with leukocytes desquamated epithelial cells fat cells and in rare instances cholesterol

**Symptoms**—The onset is slow and symptomless as a rule. In a mucocele of the ethmoid and frontal sinus frequently the first thing to be noticed is a swelling at the inner and upper angle of the orbit which feels hard and bony until the bone becomes distended and softened when a 'parchment like' crackling feel is present. Garretson<sup>1</sup> states: "When the dilated or expanded bony walls have been absorbed the swelling is definitely elastic or fluctuant. There is no tenderness or pain on manipulation. The skin is not adherent to the underlying structures and is not altered in character and moves freely over the subcutaneous tissues. Pressure over the swelling may not alter its size. The tumor like mass may remain practically stationary for years. However when the bony covering has been absorbed the condition progresses more rapidly and symptoms of pressure on the orbital contents may be noted."

"In the classical picture of this disease the eyeball is displaced downward forward and outward. If the mucocele is of a large size proptosis may be marked. Diplopia is manifested as soon as much pressure is made against the orbital contents. In certain cases a high degree of displacement occurs without diplopia. In others diplopia is one of the earliest signs. The movements of the eyeball are usually unaffected as the tumor mass is outside of the orbital contents and in fact under the periostrum of the roof of the orbit. If the mucocele is ethmoidal the swelling is usually lower down and may displace the lacrimal apparatus. This often leads to mistakes in diagnosis. The chief point of difference however is the presence of pus coming from the canaliculus when pressure is made over the lacrimal sac or possibly the presence of pus or mucopus in the nose as a result of the discharge through the nasolacrimal duct. In the latter case the swelling is usually below the internal canthal ligament of the eyelids while in case of mucocele and frontal sinus the swelling is above this ligament."

"Symptoms may be entirely wanting until the external swelling appears. Slight alteration of the visual axis as a result of displacement of the eyeball and orbital contents may give rise to diplopia. Sometimes headaches are complained of and in others only a brow ache and in others there may be a generalized uneasiness or headache."

**Differential Diagnosis**—The differential diagnosis should be made from cystic dilation of the lacrimal sac tumors of the orbit and frontal sinus dermoid cysts of the inner canthus and meningoceles. If a mucocele becomes infected the resultant pyocele gives evidence of marked inflammation with tenderness fever etc

**Treatment**—Treatment should be directed toward establishing a large permanent communication with the nose. If the mucocoele is confined to the ethmoid an intranasal operation is sufficient. Removal of the middle turbinate is usually necessary. A large opening into the mucocoele should be made.

When the mucocoele involves the frontal sinus an external operation on the frontal sinus is necessary with the removal of the floor of the frontal sinus creating a large opening into the nasal cavity.

### PNEUMOCELE

A pneumocoele (pneumatocoele) is a collection of air under pressure in the tissues. It usually escapes from a defect in the bony wall of the frontal sinus and collects adjacent to the sinus. If on the forehead an external pneumocoele results. If the defect is in the posterior wall an internal or intracranial pneumocoele is present.

A pneumocoele may follow a fracture trauma operation congenital cleft dehiscence or necrosis of the bone. The latter may be due to syphilis osteomyelitis sinusitis etc. Cases have been reported as secondary to or associated with an osteoma.

The mucous membrane or periosteum is intact over the bony defect so that a ballooning of the mucosa or periosteum occurs forming an air sac when under pressure from blowing the nose coughing etc. A pneumocoele may occur in connection with a mucocoele if air takes the place of the fluid contents.

In addition to the external and internal pneumocoeles a third type characterized by an excessive dilatation of the sinus (pneumosinus dilatans) may occur.

The dilatation of the sinus is usually associated with acromegaly localized cystitis or following fractures in the region of the sinuses. The enlargement of the sinus is more apt to result if the initiating bone changes occur before the sinuses are fully developed. Any of the sinuses may be involved either on one or both sides. The exact mechanism by which the dilatation occurs is not understood.

### OBLITERATIVE FRONTAL SINUSITIS

Obliterative frontal sinusitis according to S. R. Skillern<sup>1</sup> is an osteogenic thickening from a pathologic stimulus as the result of a protective mechanism to the dispersion of infection. It depends on the ability of the compact bone to respond to a bacterial or traumatic stimulus. It affects chiefly the anterior plate of one frontal sinus. It may be diagnosed erroneously from the roentgenogram as a failure in development of the sinus on the affected side.

Failure to make a diagnosis may result in numerous nasal operations without giving relief from the symptoms. When the roentgenogram shows a large unilateral frontal cell ending abruptly at the midline of the forehead its fellow should be suspected of osteogenesis.

According to Skillern radical operation above the superciliary line is the operation of choice

### THE SINUSES AS FOCI OF INFECTION

The part played by the sinuses as a focus of infection is less important than that of the tonsil and the symptoms are less severe

With the exception of the ethmoid the accessory sinuses seldom act as an important focus of infection and the more chronic the infection and the more evident the purulent discharge the less likelihood of their being a source of general toxemia The presence of a blood stained watery secretion is much more indicative of a systemic absorption of toxins or bacteria

According to Daland The internist suspects chronic sinusitis in adults when a disease occurs that may be due to a focus of infection when there is a history of diphtheria scarlet fever recurring influenza or rhinitis when leukopenia lymphocytosis and diminution of polymorphonuclear cells is present when no focus of infection exists elsewhere or when infected tonsils are present because infected tonsils and sinuses frequently coexist when postnasal discharge usually mucopurulent occurs each morning and when cultures from the sinus show pathogenic bacteria

Experimental evidence shows that highly toxic substances may be inserted into the sinuses without great danger of absorption even when inflammatory changes have occurred The conclusion would be that bacterial toxins from infected sinuses are likewise absorbed with difficulty

### ARGYRIA

**Synonyms** — Argyrism argyrosis

In this condition a slate-gray or bluish discoloration of the skin mucous membrane deeper tissues and organs occurs from a deposit of an insoluble albuminate of silver from the long-continued local instillation or ingestion of a soluble silver salt Argyria may occur as a localized or generalized manifestation The generalized form usually follows internal medication The localized usually follows local applications especially if denuded portions of the skin or mucous membrane are present This form is also seen in silver metal workers (occupational argyria)

As a rule several months or years are required to produce an argyria depending on the amount and frequency of the silver preparation used

The differential diagnosis should be made from hemochromatosis ochronosis and Addison's disease The staining is usually permanent however in the localized form it may diminish or even disappear spontaneously

The diagnosis in doubtful cases is made by biopsy in which the deposits of silver are found in granules or strands

The treatment consists in the permanent discontinuance of any form

of silver Urotropin by mouth has been tried for decolorization with fair results Prolonged exposure to strong sunlight or ultra violet rays should be avoided

### INTRACRANIAL COMPLICATIONS

The possible intracranial complications from disease of the nasal passages and sinuses are pych meningitis external and internal leptomeningitis extradural and subdural abscess dural fistula the various types of brain abscesses and septic thrombosis of the cavernous or the superior longitudinal sinus The other venous sinuses are rarely involved from infections of the nasal sinuses

Acute infections of the sinuses are more apt to result in intracranial complications than are chronic infections These complications are more common in males than in females (4 to 1)

All infected sinuses may give rise to an intracranial complication but an extension from a maxillary sinusitis is rare Courville and Rosenfeld state a maxillary sinusitis of dental origin is more apt to provoke intracranial suppurative lesions than any other type

Meningitis which has its origin in sinusitis is more frequently observed than thrombosis of the venous sinuses

Infections from the nose or sinuses may invade the intracranial structures from trauma through congenital dehiscences or non-closure of fetal defects by a direct pathway through the sinus wall along the sheaths of the olfactory nerves by way of the communicating veins by means of septic thrombi along the diploetic veins with a retrograde thrombophlebitis or periphlebitis to the cavernous sinus or by way of the angular or ethmoid veins to the cavernous sinus and by way of the orbit There has been some question as to the possibility of a direct extension of an infection of the sinuses to the intracranial structures by way of the lymphatic vessels However Kramer and Som\* report a case in which the infection spread from a sphenoiditis to the dura by way of the perivascular lymph spaces

### BRONCHIECTASIS

The incidence of chronic sinus disease in bronchiectasis varies with different investigators from 55 to 100 per cent The factors that would indicate the sinuses are not the chief etiologic agent are (1) The early age of onset of the bronchiectasis that is during infancy and early childhood when chronic sinusitis is not so prevalent as later in life (2) the frequency with which bronchiectasis is preceded by an acute infection involving the bronchi or alveoli (3) the tendency of the lesions to be unilateral and preponderately on the right side

The facts which would favor the theory that the sinuses are important etiologic factors are (1) The high percentage of patients with bronchiectasis in which sinusitis is present (2) the known tendency of sinus-

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\* Arch Otolaryngol 32 44 (October) 1940

itis to produce and prolong infections of the lower respiratory tract (3) the preponderate involvement of the lower lobes in bronchiectasis which would be expected if due to aspiration or gravitation of infected material from the nasal passages (4) the possibility that long-continued flooding of the lymph channels from the sinuses produces a bronchiectasis Mullin injected ink into the maxillary sinuses of rabbits and recovered the ink from the bronchial glands

It is possible that there may be a simultaneous onset of sinus and chest symptoms

Inasmuch as the sinus may be an etiologic factor of importance and in any event a contributing agent that very likely would prolong or enhance the chest pathology it is essential that the rhinologist give attention to the sinuses in the hope of giving some relief to the bronchiectasis

Mullin<sup>1</sup> believes the hyperplastic type of nasal and sinus disease is most important in the causation of chronic chest infections. The mucous membrane becomes thickened and boggy and may show polypoid degeneration. There is blocked drainage from all the sinuses with absorption of bacteria and bacterial products through the lymph channels which eventually leads to chronic peribronchial glandular enlargement. The antrum is the most important in the production of chest pathology because it is developed early, is the largest of the sinuses and is frequently infected

## PART II

# THE PHARYNX AND TONSILS

### CHAPTER XVII

## SURGICAL ANATOMY OF THE PHARYNX AND TONSILS

### SURGICAL ANATOMY OF THE PHARYNX

The pharynx is divided into the nasopharynx or that portion of the pharynx situated above the soft palate the oropharynx the portion between the soft palate and the hyoid bone and the laryngo-pharynx which extends from the hyoid bone to the lower border of the cricoid cartilage

**Nasopharynx** — The nasopharynx is formed above by the body of the sphenoid and the basilar process of the occipital bone anteriorly by the choanae and the soft palate posteriorly by the cervical vertebrae and inferiorly the nasopharynx is continuous with the oropharynx. Situated on the lateral walls of the nasopharynx behind the posterior ends of the inferior turbinates are the orifices of the eustachian tubes. Above and behind the eustachian orifice is an elevation or ridge formed by the eustachian cartilage called the eustachian cushion. Extending downward from the posterior end of the ridge is a strong fold of mucous membrane the salpingo-pharyngeal. A less prominent fold of mucous membrane the salpingo-palatine extends downward in front of the eustachian orifice. The deep pocket formed at the angle of the pharynx between the posterior ridge of the eustachian cartilage and the posterior wall is known as the fossa of Rosenmüller. Frequently adenoid tissue is found around the orifice of the tube (tubal tonsil). The roof (fornix pharyngis) and the posterior wall of the nasopharynx are the seat of lymphoid tissue (adenoid pharyngeal tonsil Luschka's tonsil) which frequently attains considerable size especially in children. The nasopharynx is covered with pseudostratified ciliated columnar (respiratory) epithelium. The oropharynx is lined with stratified squamous epithelium.

**The Adenoid** — The pharyngeal tonsil or adenoid is a lobulated lymphoid mass composed of lymphoid tissue similar to the tonsil. Its lobules or segments are arranged in regular order like separated segments of an orange with clefts or pockets between. These lobules are arranged around a central depression called the *bursa pharyngea*. Many pockets and crevices are thus presented but no compound crypts.

The distribution of adenoid tissue in the nasopharynx is chiefly on the upper and posterior walls though it may extend to the fossæ of Rosenmüller and to the orifices of the eustachian tubes. An adenoid is composed of lymphoid tissue enmeshed in a definite though compara-

tively delicate reticulum of fibrous connective tissue. The essential pathology of an adenoid consists in the hyperplasia of the lymphoid tissue of the nasopharynx which is normally present there.

The adenoid acts as a peripherally placed lymph node from which efferent ducts pass to the nearest node in the cervical chain.

The adenoid tissue consists of a fibrous connective-tissue framework supporting masses of lymphoid cells, but owing to its peripheral position it differs from the more deeply placed lymphatic nodes in having an epithelial covering upon its free surface. The supporting framework consists of fibrous septa passing through the substance of the gland from which a very delicate connective-tissue network ramifies in all directions toward the surface. It carries in it the blood vessels and the lymphatics while here and there lying in clusters in the septa may be seen many mucous glands whose ducts open on the surface. Mucous glands are frequently found within the adenoid at its base. In the meshes of the delicate network lie masses of leucocytes or lymphoid cells constituting the lymphoid tissue which forms the main bulk of the adenoids. Groups of these cells are specially differentiated in the form of more or less rounded or oval shaped areas having centers of a pale appearance while their margins are more darkly colored. These areas are the follicles or germ centers of Gooden.

**The Epithelium**—Completely covering the free surface of the adenoid and dipping down into its recesses and crypts is a layer of pseudo-stratified ciliated columnar epithelium continuous with that lining the respiratory part of the interior of the nose and the adjacent mucous membrane of the nasopharynx. The epithelium consists of more than one layer of cells, the superficial ciliated cells being columnar in type while the deeper cells forming two or three layers are smaller and rest upon a well-defined basement membrane. The epithelium covering the adenoid has no submucosa as the latter passes directly beneath the base of the adenoid. While preserving its ciliated columnar type the thickness of the epithelium varies in parts so that the lining of some of the crypts presents an irregular outline and is thinner.

**Oropharynx**—The oropharynx opens into the oral cavity at the anterior pillar of the fauces.

The soft palate (velum palati) consists of muscle fibers supported by a fibrous tissue and an outer covering of mucous membrane. A median ridge or raphé divides it into two halves. The conical shaped central projection is known as the uvula. The lateral margins of the palate on each side divide into the anterior and posterior pillars of the fauces. The anterior pillar contains the palatoglossus muscle. The posterior pillar contains the palatopharyngeus muscle. Between the two pillars is the recess in which the faucial tonsil is lodged.

The plica triangularis (tonsillaris) is a thin fold of mucous membrane stretching backward from the anterior pillar and covering a portion of the anterior surface of the tonsil.

The plica semilunaris (supratonsillaris) is the upper fold of mucous membrane which unites the two pillars at their junction.



The supratonsillar fossa is a recess of variable size situated above the tonsil and between the anterior and posterior pillars. It is formed embryologically from the second branchial cleft.

**The Lingual Tonsil** —The lingual tonsil, a sessile structure, is situated on the base of the tongue between the faucial tonsils and extends antero-posteriorly from the circumvallate papillae to the epiglottis. It is separated from the musculature of the tongue by a layer of fibrous tissue. The lingual tonsil is divided in the median line by the median glosso-epiglottic ligament. The tonsil consists of numerous rounded or circular crater-like elevations which are composed of lymphoid tissue which at their circumference are surrounded by connective tissue. In the center of each crater the mouth of the duct of a mucous gland opens. The crater or crypt is lined by stratified pavement epithelium. Branching of the crypts does not occur. They are simple tubes. The veins are part of a venous plexus which lies on the base of the tongue. Varicosities of these veins may occur. The arterial supply is from the external carotid through the dorsal lingual branch of the lingual artery. The lymphatic drainage is to the suprahyoid, submaxillary and deep cervical lymph nodes. The nerve supply is from the ninth and the superior laryngeal branch of the tenth cranial nerves.

The pharyngeal, faucial and lingual tonsils form the so-called *Waldeyer's ring*.

**Laryngo-pharynx** —The laryngo-pharynx is partially separated from the oropharynx by the pharyngo-epiglottic fold which extends from the epiglottis to the side of the pharynx.

The mucous membrane of the laryngo-pharynx is covered with pseudo-stratified ciliated columnar epithelium except over the laryngeal surface of the epiglottis, the anterior surface of the arytenoids and the free edges of the true vocal cords where stratified squamous epithelium is found. Many mucous glands and much lymphoid tissue are present. This lymphoid tissue is collected into small masses (lymphoid follicles) at numerous points throughout the pharynx.

The muscles of the pharynx consist of the three constrictors of the pharynx, the superior, middle and the inferior, the stylopharyngeus and the palatopharyngeus.

The muscles of the soft palate are the *azygos uvulae*, *levator palati*, *tensor palati*, *palatoglossus* and *palatopharyngeus*.

## SURGICAL ANATOMY OF THE TONSIL

**Embryology** —The tonsil is situated in the sinus tonsillaris between the faucial pillars and has its origin in an invagination of the hypoblasts at this point. Later the depression thus formed is subdivided into several compartments which become the permanent crypts of the tonsil. Lymphoid tissue is deposited around the crypts and thus the tonsillar mass is built up. The inner or exposed surface including the cryptic depressions is covered with mucous membrane while the outer or hidden surface is covered by a fibrous sheath called the capsule.

The anlage of the tonsils appears in early fetal life. They are visible in the fourth month, at first as simple invaginations of the mucous membrane at a point between the second and third branchial arches at the second branchial pouch.

According to Landois and Stirling, the development of the faucial tonsil is most easily studied in the rabbit, where the single primary crypt generally remains without branches through life, and there the tonsil first appears in embryos  $\frac{3}{8}$  inch long (occipito-sacral measurement), or at about twelve days as a shallow epithelial fold whose apex points directly backward into the connective tissue concentrically condensed around the pharynx. At this stage there is no infiltration of the leukocytes in the connective tissue around the crypt, and it is not until the embryos are about twenty-one days old ( $1\frac{1}{16}$  inches long) that the leukocytic infiltration becomes evident. The crypt has then become much deeper and broader, and by its ingrowth has produced a condensation of the connective tissues at right angles to the original peripharyngeal condensation, as well as a great increase in the number of capillary blood-vessels. From this stage the elongation of the crypt, the condensation of the connective tissue, the increase in the number of blood-vessels, and the amount of leukocytic infiltration go on *pari passu* until the adult condition is reached. As soon as the leukocytes appear in numbers in the submucous tissue, they proceed to escape through the epithelium, as Stohr has described.

In the fetus of the pig, the condensation of the connective tissue, especially at the apex of the tonsillar crypts, and the consequent massing of leukocytes, mainly at these points, is particularly well shown.

In the human fetus the process is the same, though complicated by the early ramification of the original epithelial crypt and the appearance of fresh ones. The crypts become so deep that the cells from the surface layers of their epithelium cannot at once be thrown off into the mouth, and remain as a concentrically arranged mass of degenerated hornified cells filling up the lumen of the crypt, this mass is ultimately forced out by the *vis a tergo* of the leukocytes emigrating through the epithelium. It will at once be seen how closely this resembles the formation of the concentric corpuscles of the thymus.

The prime factor in the formation of the tonsils is the epithelial ingrowth, which partly mechanically compresses the meshes of the connective tissue, and partly causes proliferation of the connective cells and vessels by the slight irritation it produces, thereby making it easier for the leukocytes to escape from the thin-walled capillaries and veno-capillaries so formed, and, when they have escaped, causing them to be detained in the finely meshed connective tissue longer than in other situations. As the leukocytes are well supplied with nutriment, they divide by mitosis in large numbers, as Flemming and his pupils first showed, and at a late stage in development (with great variations in individuals), "germ centers" are formed, where a special arrangement of connective tissue and vessels favors the process of division.

The lingual and pharyngeal tonsils develop in the same way as the

faucial. His shows that all the tonsils arise behind the membrana pharyngis and consequently all these epithelial ingrowths pass into connective tissue already condensed around the primitive alimentary canal.

**Anatomy** — The faucial tonsils, one on each side of the oropharynx, are almond-shaped masses of lymphoid tissue imbedded in an apparent fibrous capsule. The inner or free surface is covered by a closely adherent stratified squamous epithelial membrane. This epithelium extends into the blind pouches or crypts which have their openings onto the surface of the tonsil. The epithelium lining the crypts is very thin and offers poor protection to bacterial infection.

The tonsil does not always completely fill the sinus tonsillaris, the unoccupied space above it being known as the supratonsillar fossa.

The outer aspect of the tonsil is loosely attached to the superior constrictor muscle of the pharynx, thus subjecting it to compression with every act of deglutition. The palatoglossus and palatopharyngeus muscles of the pillars also compress the tonsil.

As viewed under the microscope the tonsil consists of three chief elements: The connective tissue, the germinating follicles and the interfollicular tissue.

1 The connective tissue, that is the trabecula or reticulum, acts as a supporting framework to the tonsil substance proper. The trabeculae carry blood vessels, nerves and lymphatics.

2 The germinating follicles (Fig. 172) are the centers wherein the larger mother cells of the leukocytic group undergo karyokinesis and form young lymphoid cells.

3 The interfollicular tissue is made up of lymphoid cells in various stages of development. The cells making up this interfollicular tissue differ in size and shape according to their location. They are greater in number around the follicles and show greater difference in their anatomic construction in the immediate neighborhood of the crypts.

**Capsule** — The tonsil is always described as having a capsule, but the existence of a definite capsule is denied by certain anatomists. What for all clinical purposes serves as a capsule is a white fibrous sheath called the pharyngeal fascia that encloses four fifths of the tonsil.

Fowler and Todd<sup>1</sup> have found in their dissections a thin delicate areolar tissue separating the true tonsillar tissue from the muscles of its bed. This areolar tissue can be split into layers between which are spaces of varying size. This would account for the ease of dissecting the upper pole of the tonsil and also for the tendency of a peritonsillar abscess to burrow around the upper half of the tonsil.

The capsule of the tonsil sends out trabeculae which pass into the parenchyma. These trabeculae carry blood vessels, nerves and efferent lymphatics. Afferent lymphatics are absent.

**Plica Triangularis** — The plica triangularis is a normal structure appearing in embryonal life and in some of the lower animals develops

<sup>1</sup> Fowler and Todd. Jour. Am. Med. Assn. 90:20, 1928.

into the tonsil itself. There is no muscular tissue in the *plexus triangularis* and it should be removed with the tonsil. When it is left in place it may form a pocket or pouch where food and other debris collect. It is the source of considerable local irritation or the lymphoid tissue with which it is thickly studded may be the seat of a future hyperplasia or infection.

**Crypts**—The crypts from eight to twenty in number are usually tubular and almost invariably extend the entire depth of the tonsil to the capsule on its outer surface. Most are compound and they divide below the surface into two or more tubules. They are usually comparatively straight though they may be tortuous in their course. Those opening in the supratonsillar fossa usually extend downward and outward whereas in the lower portion of the tonsil their direction is outward.

The subepithelial connective tissue which is present in a marked degree beneath the surface epithelium disappears as soon as the epithelium starts to form the crypts. This permits the epithelial cells to come in direct contact with the lymphatic structures of the tonsil and very frequently it is impossible to distinguish a dividing line between the epithelium of the crypt and the interfollicular tissue. The epithelium of the crypt, unlike its progenitor which covers the surface of the tonsil does not form a compact unbroken barrier of protection. For the greater part of its extent it presents an intact line only one or two possibly three cells in thickness. Toward the parenchyma the epithelial cells show a peculiar condition. They are separated from each other by interposed cells varying in type from slightly changed epithelial cells to well formed lymphocytes. The epithelial cells may also extend from the crypt into the tonsillar substance suggesting the ramifications of a malignant epithelioma. The smaller terminal invaginations of the cryptal epithelium are usually solid sprouts frequently with central keratinized cores. The lumen of the crypt is formed by the subsequent exfoliation of the keratinized cells.

Clinically the crypts seem to be the source of the greatest amount of local and constitutional disturbances as they often become filled with food tissue debris and bacteria.

**Tonsillar Fossa (Sinus Tonsillaris)**—The anterior pillar contains the palatoglossus muscle (Fig. 161) and forms the anterior boundary whereas the posterior pillar contains the palatopharyngeus muscle and forms the posterior boundary of the sinus.

The palatoglossus has a fan shaped origin in the oral surface of the soft palate and terminates in the lateral side of the tongue.

The palatopharyngeus is a vertically arranged muscle attached above to the soft palate the eustachian tube and the base of the skull. It extends downward to the upper esophageal wall. This muscle is of greater importance than the palatoglossus. Great care should be taken not to injure this muscle during the tonsil operation.

The pillars meet above to unite with the soft palate. Inferiorly they diverge and enter into the tissues at the base of the tongue and the

lateral wall of the pharynx respectively. The outer wall of the tonsillar fossa is composed of the superior constrictor muscle of the pharynx.

The superior constrictor muscle has transversely disposed fibers. It forms the circular musculature of the pharynx. It originates from the lower portion of the internal pterygoid plate, the hamular process, the pterygomandibular ligament and the mandible.



FIG. 161.—In front of the raphe is seen the buccinator muscle. The superior constrictor muscle runs back from the raphe. Below these from without inward are seen the mandible, internal pterygoid, lingual nerve, styloglossus, glossopharyngeal nerve, stylopharyngeus and palato-pharyngeus, behind which is seen the tonsil capsule (its upper lobe a hood, its lower lobe a pocket). Behind and lateral to these are vertical fibers of the pharynx running to the soft palate and the hamular process. These are continued posteriorly as coarser fibers—the palatopharyngeus. Vessels and nerves are shown descending from the palate to the tonsil together with the attachment of the tonsillopharyngeus in the groove between the upper and lower lobes. 1 Levator palati, 2 palatopharyngeus, 3 superior constrictor, 4 palatoglossus, 5 pterygomandibular raphe with superior constrictor medially and buccinator laterally, 6 upper lobe or hood of tonsil (Fowler and Todd Jour. Am. Med. Assn.).

Fowler and Todd<sup>1</sup> describe a fourth muscle which they named the tonsillopharyngeus. It is formed by fibers of the lateral part of the palatopharyngeus. It is attached to the capsule of the tonsil at the junction of the upper and lower lobes.

<sup>1</sup> Jour. Am. Med. Assn. 90:20, 1928.

**Lymphatics.**—The lymph nodes are subdivided into groups according to the region occupied. The suboccipital group (two to three) usually lies near the insertion of the occipital muscle. They receive afferent vessels from the lower occipital part of the head. The efferent vessels run to the upper substernal mastoid nodes. The mastoid or retroauricular nodes (usually in pairs) are found near the insertion of the sterno-cleido-mastoid muscle. They receive afferent vessels from the temporal portion of the head, the internal surface of the ear, and from the posterior part of the auditory canal.

The parotid nodes (three to sixteen) are composed of the superficial and deep nodes over the parotid area, under the superior aponeurosis. The afferent vessels are received from the external part of the ear, the skin over the temporal and frontal regions, eyelids, outer part of the nose and anterior part of the auditory canal.

The submaxillary group (three to six) is found along the inferior border of the maxilla lying upon the submaxillary gland. They receive afferent vessels from the nose, cheek, upper lip, external part of the lower lip, gum and anterior part of the tongue. The efferent vessels empty into the deep cervical chain.

The submental group (one to four) have afferent vessels from the skin over the chin, middle of the lower lip, floor of the mouth and the tip of the tongue. Efferent vessels go to the submaxillary nodes and to a node on the external jugular vein.

The retropharyngeal nodes (one to ten) are located on the posterior and lateral pharyngeal walls about the level of the atlas bone. The afferent vessels are from the mucosa of the nasal fossa, sinuses, nasopharynx, eustachian tube and lymphatics from the internal ear. Efferent vessels empty into the internal jugular chain.

The descending deep cervical chain (carotid group) of nodes accompanies the great vessels of the neck to the chest. They lie beneath the sterno-cleido-mastoid muscle and form the most important group of lymph nodes in the body. There are from fifteen to thirty nodes in this group. An external jugular group is placed posteriorly and externally to the internal group. The internal nodes lie on and parallel to the internal jugular.

The external group receives efferent vessels from the mastoid nodes, the suboccipital and some vessels from the nodes about the external jugular, a portion of the occipital region of the scalp, lobe of the ear, the cutaneous lymphatics from the upper part of the neck and part of the nasal fossæ and the nasopharynx.

The internal group receives efferent vessels from the retropharyngeal, parotid, submaxillary and submental nodes, a large part of the lymphatics from the tongue and nasopharynx, all of the middle and inferior part of the pharynx, the cervical part of the esophagus and trachea, nasal fossa, thyroid gland, tonsils, hard palate, orbit, and larynx.

The tonsillar lymphatic vessels drain into the deep cervical chain underneath the sterno-cleido-mastoid muscle, thence to the thoracic nodes, and finally into the thoracic duct. By this route, infection may

be carried to all parts of the body. The tonsil under certain conditions being peculiarly susceptible to infection becomes therefore the atrium of infection for a great variety of diseases extraneous to itself.

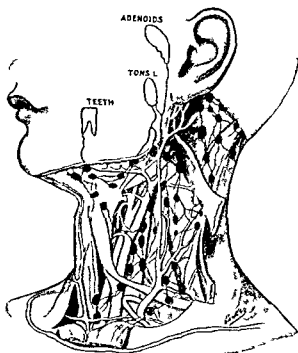


FIG. 167.—The lymphatic nodes and vessels of the neck which drain the teeth, tonsils, adenoid, pharynx and mastoid region.

In reference to the tonsil as the portal of infection in tuberculous processes it is generally admitted that this often takes place through the tonsil and extends thence through the lymphatics of the deep cervical chain on into the thorax. It then passes through the hilus of the lung into the visceral pulmonary lymphatics.

**Blood Supply**—The blood supply to the pharynx comes from many sources and is sometimes irregular. The chief supply is derived from the ascending pharyngeal and faucial branches of the external carotid and from the superior palatine of the internal maxillary.

The tonsillar artery (Fig. 208) a branch of the external maxillary is the chief vessel to the tonsil though the ascending palatine another branch of the external maxillary sometimes takes its place. The tonsillar artery passes upward on the outer side of the superior constrictor muscle through which it passes and gives off branches to the tonsil and soft palate. The ascending palatine another branch of the external

maxillary, also sends branches through the superior constrictor muscle to the tonsil. The ascending pharyngeal passes upward outside of the superior constrictor, and when the ascending palatine artery is small, it gives off a tonsillar branch which is correspondingly larger. The *dorsalis linguæ*, a branch of the lingual artery, ascends to the base of the tongue and sends branches to the tonsil and pillars of the fauces. The descending or posterior palatine artery, a branch of the internal maxillary, supplies the tonsil and soft palate from above, forming anastomoses with the ascending palatine. The small meningeal artery sends more branches to the tonsils, though they are of minor importance.



## CHAPTER XVIII

### DISEASES OF THE PHARYNX AND NAUSES

#### SIMPLE ACUTE PHARYNGITIS

This form of acute pharyngitis is usually accompanied by acute rhinitis or cold though the pharynx may be involved to a greater extent than the nose or larynx.

**Etiology and Pathology**—The etiology and pathology is the same as that of acute rhinitis.

**Symptoms**—The onset is characterized by malaise and a slight rise in temperature as in acute rhinitis. The borders of the soft palate and uvula are slightly red while the adjacent mucous membrane is normal in appearance. As the disease progresses the uvula becomes slightly edematous and the secretions are increased. It may become markedly edematous and painful though this is not common. The tonsils are usually congested. Pain upon swallowing stiffness and aching of the muscles of the neck are noted.

**Treatment**—The general treatment as given for acute rhinitis should be given. As the acute affection is occasionally dependent upon the presence of chronic rhinitis and sinusitis these conditions should receive appropriate attention.

Local treatment should vary according to the stage of the inflammation. Antiseptics such as merthiolate 1:1000 metaphen 1:2000 etc may be applied as gargles sprays paints or lozenges. Gargles are suited to inflammations of the soft palate uvula and anterior pillars of the fauces. Sprays and paints are especially good methods of making applications to the pharynx as a gargle seldom reaches this area.

Insufflation of one of the powdered sulfonamides alone or in combination into the nasal cavity and pharynx is one of the most effective methods of using local chemotherapy. The powder seems to be more effective than the various suspensions or solutions of the sulfonamides. A solution of penicillin (200 to 1000 units per cc) in normal saline may prove to be equally satisfactory.

The inhalation of steam with the compound tincture of benzoin added 1 tablespoonful to the pint of boiling water should be used if the throat is painful.

#### EDEMA OF THE UVULA

Acute inflammation of the faucial structures is frequently attended by edema of the uvula (Fig 163). It frequently follows acute infections as well as excessive or faulty use of the voice.

It usually disappears spontaneously. If troublesome the edematous portion may be scarified by multiple punctures which allow the excess serum to escape.

Astringent lozenges will be found efficacious in giving comfort to the patient

### INFLUENZA

Influenza is an acute infection of the respiratory tract occurring in epidemics of varying severity. It seems to be due to two distinct types of virus types A and B and possibly others as yet unidentified. Type A is responsible for the great majority of cases.



FIG. 163. Edema of the larynx.

Epidemics are characterized as a rule by their sudden appearance, rapid spread, low mortality, and prompt subsidence. The epidemic usually reaches its peak in from three to four weeks and subsides in from six to eight weeks after the onset. From 10 to 40 per cent of the population in the area of the epidemic may be affected. At times outbreaks with high mortality occur, as in 1918.

**Symptoms** — The initial symptoms are a mild rhinitis accompanied by a sensation of dryness or fullness of the throat, especially on swallowing.

A mild or severe headache in the frontal or occipital regions is present in almost all instances. A feeling of malaise or fatigue is marked and out of proportion to the appearance of the throat. The back and legs usually ache frequently to a marked degree.

The temperature is usually elevated from 1 to 4° F. A secondary rise several days later is not uncommon.

At times a gastro-intestinal disturbance accompanies the disorder, especially in children.

An early tickling of the throat with an unproductive cough is a prominent feature. The face is flushed and frequently an erythema over the chest is noted.

The throat and pharyngeal lymphatic structures are diffusely redened and inflamed but without spots or membrane in most instances. The tonsils may be spotted but more frequently are only inflamed similar to the pharynx.

A leukopenia or a normal leukocyte count is the rule. If a secondary infection or complication occurs a moderate leukocytosis may be present.

A mild cervical adenitis with rather marked tenderness is usual. The conjunctivae are congested or inflamed in almost all the cases in the early stage.

**Treatment**—Preventive treatment is attained by avoiding infected individuals and crowds during epidemics.

Prophylactic immunization against types A and B strains of influenza seems to have the greatest effect during the second week following the infection (Commission on Influenza<sup>1</sup>). At this time the average influenza rate was found to be reduced about 85 per cent.

The vaccine is given in one subcutaneous dose of 1 cc. with a repeat dose of 1 cc. at three months intervals during the influenza season.

Treatment is largely symptomatic in uncomplicated cases as chemotherapy does not seem to influence the primary virus infection.

The patient should be kept in bed for from twenty-four to forty-eight hours after the febrile stage has passed. Cold air admitted to the room should be reduced as an excess of cold air or draughts irritate the respiratory passages. Steam inhalations or a high moisture content of the room reduces the mucous membrane irritation and tendency to cough. If the patient loses sleep from the cough some form of cough mixture with codeine is indicated. Mild antipyretics, laxatives and enemata should be given as needed. A fluid intake of 3000 cc. in adults should be taken daily.

The sulfonamides or penicillin should be given for the secondary invaders and complications as indicated but will not alter the course of the primary virus.

**Complications**—Complications are due in almost all instances to secondary bacterial invaders which seem to be activated by the virus. The hemolytic streptococcus is recovered in most ear, nose and throat complications. The mortality varies with the type, virulence and location of the secondary invaders.

## ACUTE HEMOLYTIC STREPTOCOCCIC THROAT INFECTIONS

**Synonyms**—La grippe, influenza, septic sore throat.

Throat (upper respiratory tract) infections due to the hemolytic streptococcus have assumed a role of vast importance in recent years on account of the almost unlimited variety of complications that may result as secondary manifestations. These secondary manifestations (or complications) are often difficult to follow and to correlate. That a bacteremia is responsible for many, if not a majority of these complications is becoming increasingly evident.

**Bacteriology**—The hemolytic streptococci are spherical or slightly oval. They form definite, often quite long chains. Occasionally they are seen in pairs or in short chains. Their hemolytic activity is caused

by a hemolysin called streptolysin, which gives rise to an antibody (Besredka). In severe infections the hemolysis may occur in the vessels with the resulting hemoglobinemia.

There are many classifications of the streptococcic family. Most investigators have followed Schottmüller in discarding the old term *Streptococcus pyogenes* for the terms *Streptococcus hemolyticus*, *Streptococcus viridans* and *Streptococcus mucosus*, on the basis of their reactions and appearances on blood mediums.

Gordon<sup>1</sup> classifies the streptococci according to their ability to hemolyze blood and to ferment sugars, raffinose and mannite. He recognizes three main groups.

	Hemolysis	Raffinose	Mannite
1. <i>Streptococcus pyogenes</i> or hemolyticus	+	-	-
2. <i>Streptococcus salivarius</i> or viridans	-	+	-
3. <i>Streptococcus fecalis</i> or enterococcus	-	-	+

Holman,<sup>2</sup> in promulgating his general classifications of the streptococcus family, found that the great majority of his hemolytic strains fell into one group but was able to differentiate seven variant groups in addition. Kinsella and Swift<sup>3</sup> found twenty-eight hemolytic strains yielded such marked cross-fixation as to lead to the conclusion that all were closely related or practically identical. Birkhaug<sup>4</sup> differentiated three groups of the hemolytic streptococcus, a group causing erysipelas, a group causing scarlet fever and a large series of miscellaneous hemolytic streptococci producing a variety of pyogenic infections. Dochez, Avery and Lancefield<sup>5</sup> demonstrated four major groups of the hemolytic streptococci on a basis of the degree and character of hemolysis produced.

Most observers recognize the following groups:

1. Beta type ( $\beta$ ) which produces a gray biconvex or disk-like colony surrounded by a complete and clearly-defined zone of hemolysis 2 to 4 mm in diameter, with no greenish or yellowish discoloration of the medium and on microscopic examination no intact cells remain in the hemolyzed zone about the colony.

2. Alpha prime type ( $\alpha'$ ), which is similar to the beta type, but under the microscope a few corpuscles are seen to remain throughout the hemolyzed zone. These incompletely hemolyzed corpuscles are more numerous next to the colony.

3. Alpha type ( $\alpha$ ), which is a green-producing variety with no hemolysis.

4. Gamma type ( $\gamma$ ), which forms a gray color with no hemolysis.

Of the four groups the beta type is the one with which we are primarily concerned, as the other types are apparently not so virulently pathologic.

<sup>1</sup> Gordon, M. H. Classification of Streptococci, Jour. State Med., 30, 432, 1922.

<sup>2</sup> Holman, W. L. Streptococci, Jour. Med. Res., 34, 377, 1916.

<sup>3</sup> Kinsella, R. A., and Swift, H. F. Classification of Hemolytic Streptococci, Jour. Exper. Med., 28, 169, 1918.

<sup>4</sup> Birkhaug, Konrad E. Studies on the Biology of the Streptococcus Erysipelatis Bull. Johns Hopkins Hosp., 37, 85, 1925.

<sup>5</sup> Dochez, A. R., Avery, O. T., and Lancefield, R. C. Biology of Streptococcus, Jour. Exper. Med., 30, 179, 1919.

for man. Bloomfield and Felty<sup>1</sup> in a study of 1000 cultures of the hemolytic streptococcus were unable to recover from a case of tonsillitis erysipelas scarlet fever or other acute streptococcic infections a strain which did not have typical and undoubted beta type characteristics. They insist the alpha prime alpha and gamma types are non pathogenic for man. The hemolytic activity of this organism is undoubtedly an indicator of the pathogenicity.

Dochez<sup>2</sup> Gordon<sup>3</sup> and others have shown that the type of beta hemolytic streptococcus found in scarlatinal angina is in general a specific type distinguishable from the types of *Streptococcus hemolyticus* causing other kinds of angina and septic conditions in general. Bliss<sup>4</sup> has shown that this streptococcus scarlatina can be recovered from all patients with scarlet fever as well as from contacts and healthy carriers.

Beckwith and Eddie<sup>5</sup> have shown that *Streptococcus hemolyticus* is very resistant to cold. They found it uninfluenced by freezing temperature but readily killed by heat.

**Epidemics**—In recent years, about a dozen or more epidemics of 'septic sore throats have been reported. Bacteriologic studies have invariably shown a streptococcus as the offending organism. Capps<sup>6</sup> described it as a capsulated hemolytic streptococcus. Davis and Rosenow<sup>7</sup> called it a peculiar streptococcus. Later Davis acknowledged its relationship to *Streptococcus hemolyticus* and thought that the two were identical.

A few of these epidemics were traced to infected milk. The Boston epidemic of 1911 was the first in this country to be so attributed. The investigations of Krumwiede and Valentine<sup>8</sup> and Davis and Capps<sup>9</sup> indicate that the animal is a secondary host having been infected originally by a human being. This infection is carried through an abrasion of the teat or udder. Davis and Capps have shown that when the hemolytic streptococcus is planted on the teat with an abrasion the infection may ascend the ducts and cause a prolonged infection in the udder. The streptococci are shed in the milk in large numbers for weeks and months.

<sup>1</sup> Bloomfield A. L. and Felty V. P. On the Dissemination of Hemolytic Streptococci Among a Group of Healthy People. *Bull. Johns Hopkins Hosp.* 134: 414, 1923.

<sup>2</sup> Dochez A. R. and Sherman L. Significance of *Streptococcus Hemolyticus* in Scarlet Fever. *Jour. Am. Med. Assn.* 82: 542, 1924.

<sup>3</sup> Gordon M. H. Serological Study of Hemolytic Streptococci. *Brit. Med. Jour.* 1: 632, 1921.

<sup>4</sup> Bliss W. P. Biology of *Streptococcus*. Antigenic Relationship Between Strains of *Streptococcus Hemolyticus* Isolated from Scarlet Fever. *Jour. Exper. Med.* 36: 55, 1920.

<sup>5</sup> Beckwith T. D. and Eddie B. U. Hemolytic Streptococci of Human Origin in Relation to Heating and Freezing in Milk Industry. *Jour. Lab. and Clin. Med.* 9: 316, 1924.

<sup>6</sup> Capps J. A. Epidemic of Sore Throat Due to Milk. *Jour. Am. Med. Assn.* 58: 1111, 1917.

<sup>7</sup> Davis D. J. and Rosenow E. C. Epidemic of Sore Throat Due to a Peculiar Streptococcus. *Jour. Am. Med. Assn.* 58: 773, 1917.

<sup>8</sup> Krumwiede and Valentine. Coll. Stud. Bureau of Lab. City of N. Y. 1914, 1915.

<sup>9</sup> Davis D. J. and Capps J. A. Experimental Bovine Mastitis Produced with Hemolytic *Streptococcus* of Human Origin. *Jour. Infect. Dis.* 15: 135, 1914.

**Symptoms**—The clinical picture of an acute throat (upper respiratory tract) infection is a mild coryza with a sensation of fulness or dryness of the pharynx with slight discomfort on swallowing. Headache frontal or occipital may be present. Sense of malaise is rather marked and out of proportion to the appearance of the throat. Chilliness and slight fever is usually present especially in children. The temperature in uncomplicated cases lasts from three to four days. A secondary rise is not uncommon. The back and legs may ache but just as frequently the aching is absent. Occasionally a slight nausea and vomiting are present in children. Hoarseness is uncommon but a slight tickling cough is usually present. The face is flushed and occasionally an erythema over the chest is found. The anterior cervical lymph nodes are usually tender and slightly swollen. The posterior lymph nodes are occasionally involved. A mild or marked conjunctivitis is present in almost 100 per cent of the cases during the acute stage.

The throat is diffusely reddened and inflamed. The uvula is edematous and hyperemic. The tongue is coated and the breath fetid. All the lymphatic structures of the mouth and pharynx are red and swollen.

The tonsils may be spotted but more frequently are only inflamed similar to the pharynx.

A moderate leukocytosis is usual persisting from four to eight days after the temperature has become normal. The red cells are not affected in uncomplicated cases.

In those cases of acute otitis media or acute throat infection in which a bacteremia is suspected the patient is usually bright-eyed and alert (mental stimulation). There is no clouding of the intellect even with a high temperature. Delirium is usually absent. The appetite is good with a desire for much water. The temperature usually has a fall and rise during the twenty-four hours. The patient feels fairly comfortable. If it is an otitic case and an operation is performed there is usually a drop in the temperature but a subsequent rise. The abscesses of a septicemia are comparatively rare in this type of a bacteremia.

Felty and Hodges<sup>1</sup> in a carefully worked up series of 40 cases of acute throat infection report obtaining practically pure cultures of the beta hemolytic streptococcus in 100 per cent of their cases during the acute stage of the infection. They believe that all cases of acute tonsillitis (pharyngeal lymphoid tissue) are due to the beta hemolytic streptococcus and the disease itself should be fixed on a firm etiologic basis with a definite entity such as erysipelas rather than a clinical syndrome which follows any bacterial infection of the pharynx. According to Felty and Hodges as well as Bloomfield<sup>2</sup> the green streptococci and Gram negative cocci represent the fixed normal flora of the mouth. This is not in accord with the usual conception of these organisms.

<sup>1</sup> Felty A. R. and Hodges A. B. Clinical Study of Acute Streptococcus Infection of Pharyngeal Lymphoid Tissue. *Bull. Johns Hopkins Hosp.* 34: 330, 1923.

<sup>2</sup> Bloomfield A. L. Significance of Bacteria Found in Throats of Healthy People. *Bull. Johns Hopkins Hosp.* 22: 33, 1921.

Felty and Hodges attribute this supposed misconception to the employment of surface cultures, instead of the poured 'shake' cultures as used by Brown.<sup>1</sup>

**Immunity.**—Certain persons possess a natural immunity to this organism, as is true in other bacterial diseases. Most people, however, have repeated attacks. Acquired immunity is of slight or short duration, as demonstrated clinically by the same person having the disease repeatedly and within a short space of time. Certain animals, such as the cow, mouse, cat and rabbit, are susceptible to the hemolytic streptococcus. Others, such as the rat, guinea-pig and birds, are immune.

**Carriers**—It may be conservatively estimated that during the winter months one-half of the population of the temperate zone are active carriers of the hemolytic streptococcus. Pilot and Davis<sup>2</sup> recovered hemolytic streptococcus from surface swabs of the tonsils in 49 per cent of the patients examined. From the crypts they obtained the organism in 59 per cent of the cases, and from the crypts of the same tonsils after removal, they obtained a positive result in 92 per cent of the cases. The actinomycete-like granules in the tonsils show hemolytic streptococcus in large numbers and in a high percentage of instances (Pilot and Davis). Bloomfield and Felty<sup>3</sup> examined a group of 200 healthy women and found about 41 per cent of them carriers of the beta hemolytic streptococcus. They determined such carriage was essentially dependent on focal tonsillar infection.

It has been demonstrated that large outbreaks of epidemic meningitis may be preceded by a great rise in the carrier rate (Bloomfield and Felty).

Nakamura<sup>4</sup> examined over 2000 extirpated tonsils and found a striking increase in the incidence of the hemolytic streptococcus during the winter months, beginning in December and lasting until the middle of May.

Smillie<sup>5</sup> found that certain persons may retain the beta hemolytic streptococcus in the throat for three or four months after an attack of the disease. Sharp, Norton and Gordon<sup>6</sup> found a carrier condition of not less than eight weeks in five of eight instances.

In tonsillectomized throats Van Dyke<sup>7</sup> found the hemolytic streptococcus in 16.4 per cent of the cases. This is about the percentages found

Monograph of Rockefeller Inst of Med Res vol 9 1919

Pilot I and Davis D J Hemolytic Streptococci in Faucial Tonsils and Their Significance as Secondary Invaders Jour Infect Dis 24 386 1910

<sup>3</sup> Bloomfield A L and Felty A R On the Dissemination of Hemolytic Streptococci Among a Group of Healthy People Bull Johns Hopkins Hosp 34 414 1923

<sup>4</sup> Nakamura T Bacteriology of Extirpated Tonsils and Its Relation to Epidemic Tonsillitis Ann Surg 79 24 1924

<sup>5</sup> Smillie W G Beta Hemolytic Streptococcus Jour Infect Dis 20 43 1917

<sup>6</sup> Sharp, W B Norton, J F and Gordon J E Sore Throat Epidemic of Unusual Type Influenza Studies Jour Infect Dis 30 372 1922

<sup>7</sup> Van Dyke H B Hemolytic Streptococci in Normal Throats After Tonsillectomy Jour Am Med Assn 74 448 1920

by Nichols and Bryan<sup>1</sup> and by Pilot and Davis.<sup>2</sup> Tongs<sup>3</sup> found a somewhat lower percentage, and Simmons and Taylor<sup>4</sup> somewhat higher.

Pilot and Pearlman<sup>5</sup> examined the naso-pharynx and adenoids of 103 children aged from five to sixteen years with no evidence of a recent inflammation or sore throat and recovered the organism from the naso-pharynx and surface of the adenoids in 55 per cent of the cases. The crypts from the same adenoids produced the organism in 61 per cent of the children. The excised tonsils from the same children showed an incidence of 95 per cent.

Davis<sup>6</sup> found the organism not commonly present in the nose unless an active infection was present. He found the surface of the pharynx relatively free from the organisms in large numbers during the absence of the inflammation. In subjects with a cold he obtained it in from 50 to 60 per cent of the cases. The normal gums do not harbor the infection in large numbers. They are rarely found on the skin if clean. They are found more frequently on the skin of people of unclean habit. Schrechter<sup>7</sup> found hemolytic streptococcus in 8 out of 89 dirty people usually from the hairy parts of the body. They are easily removed by washing. Their disappearance in the gastro-intestinal tract is equally rapid. When placed in the stomach of rabbits Davis<sup>6</sup> found the organisms dead in from two to five minutes. Valentine<sup>8</sup> in his bacteriologic study of middle-ear infections found in the acute cases in which cultures were made at the time of the incision of the ear drum and at intervals following that the beta hemolytic streptococcus was the most important organism found and was recovered in nearly all cases. The bacterial flora changed after a few days.

The incidence of the hemolytic streptococcus in four common diseases by various observers gives an average as follows:

	Cases	Per cent
Scarlet fever	337	85
Measles	1245	48
Tonsillitis	128	71
Otitis media	204	74

**Channels of Infection**—There are four main channels by which the infection may be transmitted to other portions of the body:

1 Spread by continuity of tissue or by a local thrombophlebitis as

<sup>1</sup> Nichols H J and Bryan J H. Tonsil Infection Jour Am Med Assn 71 1813 1918

<sup>2</sup> Pilot I and Davis D J. Hemolytic Streptococci in Faucal Tonsils and Their Significance as Secondary Invaders Jour Infect Dis 24 386 1910

<sup>3</sup> Tongs M S. Hemolytic Streptococci in Nose and Throat Jour Am Med Assn 73 1050 1919

<sup>4</sup> Simmons J S and Taylor A E. Bacterial Carriers in Upper Respiratory Tract Jour Am Med Assn 72 1855 1919

<sup>5</sup> Pilot, I and Pearlman S J. Bacteriological Studies of Upper Respiratory Passages Jour Infect Dis 29 47 1921

<sup>6</sup> Davis D J. Hemolytic Streptococcus with Special Reference to Their Role in Acute Respiratory Infections Jour Am Med Assn 72 319 1919

<sup>7</sup> Schachter. Proc Chicago Path Soc 10 301 1918

<sup>8</sup> Valentine E. Bacteriologic Study of Middle Ear Infections Jour Infect Dis 35 177 1924



evidenced by a peritonsillar abscess infection travelling through the tissues of the neck and even invading the mediastinum otitis media mastoiditis lateral sinus thrombosis brain abscess etc

2 Entrance into the alimentary canal via the stomach As before mentioned Davis<sup>1</sup> has shown experimentally the fate of the hemolytic streptococcus when injected into the stomach of rabbits even in large numbers They are all killed in from two to five minutes Dible<sup>2</sup> only found 2 cases of hemolytic streptococcus in 309 stools Goodby<sup>3</sup> examined the feces of 50 cases and failed to find *Streptococcus salivarius* (viridans) in any of them Apparently the streptococci do not pass the barrier of the gastric juices in any appreciable number

3 Extension by way of the lymphatics This is demonstrated in many cases by a cervical adenitis, retropharyngeal abscess etc Goodman<sup>4</sup> and Mosher<sup>5</sup> report cases of primary jugular thrombosis secondary to a throat infection which they attributed to an extension by way of the lymphatics

4 Direct infection of the blood stream or blood vessels With the hemolytic streptococcus it is highly probable that this is the most important route by which the bacteria are conveyed from the primary focus whether of the acute or chronic type

**Complications by Way of the Blood Stream**—The possibility of a primary streptococcic peritonitis secondary to a throat infection by way of the blood stream has been denied until Kunzler<sup>6</sup> reported a positive case in 1904 in which the organism was recovered from the throat peritoneal cavity and the blood Necropsy revealed no intraperitoneal lesion Somewhat later, Chapelle<sup>7</sup> reported 18 cases of rapidly fatal peritonitis in children during an epidemic of sore throat He demonstrated the organism in the throat, peritoneal cavity and the blood Rabinowitz<sup>8</sup> reports 8 cases not in epidemic form, in which he established a preceding sore throat in most cases The blood culture was positive thirty six hours after the onset of the illness in 1 of the 4 cases in which the cultures were taken Ransohoff and Greenebaum<sup>9</sup> report a case of acute hematogenous streptococcic peritonitis in a child in which the same organism was recovered from the throat peritoneal cavity and the blood MacLennan and McNee<sup>10</sup> report a case of a child

<sup>1</sup> Davis D J Hemolytic Streptococcus with Special Reference to The r Role in Acute Respiratory Infection Jour Am Med Assn 72 319 1919

<sup>2</sup> Dible J H Enterococcus and Fecal Streptococci Jour Path and Bacteriol 24 3 1921

<sup>3</sup> Goodby K W Streptococcic Infections Arising from the Mouth Jour State Med 30 415 1922

<sup>4</sup> Goodman C Jugular Thrombosis Ann Otol Rhinol and Laryngol 26 527 1917 Ibid 34 213 1925

<sup>5</sup> Mosher H P Deep Cervical Abscess and Thrombosis of Internal Jugular Ven Laryngoscope 30 365 1920

<sup>6</sup> Kunzler Munchen med Wchnschr 51 1920 1904

<sup>7</sup> Chapelle Arb a d path Inst zu Helsingfors 2 583 1907

<sup>8</sup> Rabinowitz M A Acute Hematogenous Streptococcic Peritonitis Am Jour Med Sci 157 797 1919

<sup>9</sup> Ransohoff J L and Greenebaum J V Acute Appendicitis and Pneumonia in a Baby Fourteen Months of Age Am Jour Dis Child 27 76 1924

<sup>10</sup> MacLennan and McNee Brit Jour Dis Child 10 253 1913

who had a primary hemolytic streptococcic peritonitis secondary to measles. Operation revealed a general peritonitis and a normal appendix. Pus from the abdomen and the blood culture showed the hemolytic streptococcus.

Poynton and Paine<sup>1</sup> showed that in young rabbits a micrococcus could produce a local lesion in the appendix by direct blood stream infection. Their later investigations<sup>2</sup> led them to believe that a cause of appendicitis may be a streptococcal invasion through the blood stream from a throat infection. They report a case that seems to prove conclusively that this is the case.

Rosenow's<sup>3</sup> experiments indicate that appendicitis frequently is a blood borne infection secondary to some distant focus such as the throat. This would explain the occurrence of appendicitis at times almost in epidemic form, when throat infections are quite prevalent.

Keegan<sup>4</sup> reports an epidemic of hemolytic streptococcic sore throat during February, 1919 at the U. S. Naval Hospital at Chelsea, Mass. in which attention was first called to the presence of an epidemic by the numerous instances of a postoperative rise in temperature in those who had abdominal surgical conditions. This rise in temperature was associated with a sore throat in all cases and a subsequent infection of the previously clean wounds. Hemolytic streptococcus was recovered from the throat and the wound in all cases. Blood cultures were taken at various periods in 10 cases with negative results; however the clinical evidence of a bacteremia in these cases could hardly be more strikingly indicated than by the frequent deep infection of the traumatized tissue with the hemolytic streptococcus following a mild throat infection of the same organism.

Smith<sup>5</sup> reports an epidemic of streptococcic hemolytic septicemia in a ward of 17 babies aged from one to seven and a half months secondary to a throat infection of the same organism. Seven babies died and came to necropsy. All gave a positive culture of *Streptococcus hemolyticus* from the heart's blood. Search of the literature reveals many occasions in which organisms have been recovered from the blood at necropsy when repeated cultures had failed during life.

Bayne-Jones<sup>6</sup> reports an acute respiratory disease that suddenly broke out in 25 laboratory cats. The onset was sneezing, thin discharge from the nose and conjunctivitis. All but 2 cats died in fifteen days. At necropsy all the cats presented the same general appearance, a green

<sup>1</sup> Poynton F. J. and Paine A. A Further Contribution to the Study of Rheumatism. *Lancet* ii 1189 1911.

<sup>2</sup> Poynton F. J. and Paine A. The Etiology of Appendicitis as a Result of a Blood Infection. *Lancet* vol ii 1912.

<sup>3</sup> Rosenow E. C. The Bacteriology of Appendicitis and Its Production by Intravenous Injection of Streptococci and Colon Bacilli. *Jour Infect Dis* 16 240 1915.

<sup>4</sup> Keegan J. J. Hospital Epidemics of Streptococcus Sore Throat with Surgical Complications. *Jour Am Med Assn* 72 1434 1919.

<sup>5</sup> Smith L. H. Epidemic of Streptococcus Hemolyticus Septicemia. *Am Jour Dis Child* 24 171 1922.

<sup>6</sup> Bayne-Jones S. Respiratory Infection and Septicemia of Cats Due to Hemolytic Streptococcus. *Jour Infect Dis* 31 474 1922.

mucopurulent exudate filled the cavities of the nose and extended over the naso-pharynx to the bifurcation of the trachea. The cranial sinuses were normal. No evidence gross or microscopic, could be found of involvement of other organs. The beta type hemolytic streptococcus was recovered from the throat and from plating a single drop of the heart's blood of these cats. Further experiments with this strain of bacteria showed an early loss of ability to produce the disease when placed within the nose of cats.

Brennemann<sup>1</sup> calls attention to the frequency of abdominal pain in children in throat infections and suggests the probability of a blood borne infection to the mesenteric and retroperitoneal lymph nodes. He reports 2 such cases.

In many cases of arthritis hemolytic streptococcus may be recovered from a lymph node removed from various portions of the body. Occasionally, blood from a vein contains such organisms (Crowe). This suggests that a bacteremia is not uncommon even in the absence of an acute infection.

Tanaka and Crowe<sup>2</sup> have shown by a series of carefully prepared slides that the epithelium that lines the crypts in a normal tonsil has a rich capillary blood supply. The large collecting veins that surround each crypt lie just under the basement membrane, and a destruction of this epithelium is the most common microscopic finding in chronic tonsillitis thereby affording an easy pathway for entrance of bacteria into the blood stream. This entrance during an acute infection should be doubly easy. The rather striking sequence of a cardiac involvement following an acute streptococcal throat infection is very suggestive of the possibilities of this method of disease transmission.

Herrick<sup>3</sup> has shown that epidemic meningitis is preceded by a general invasion of the blood stream, and that in some cases the infection remains a bacteremia and never localizes in the meninges or elsewhere.

Otitis Media.—Many cases of mastoidectomy performed in the first week of the otitis are reported in the literature. When the offending organism is the hemolytic streptococcus, an almost equal number of grave blood borne complications are also reported. It is logical to assume that, with this organism present these complications, in the vast majority of cases, are accounted for by an entrance of the bacteria (not necessarily a thrombophlebitis) into the blood stream. The probability of this bacteremia is greatly enhanced by too early an interference before the infected area is walled off by Nature's protective mechanism. Bacteriologic investigation as well as clinical observation tend to uphold this view. In these too early operations the very complications we are trying to prevent are rendered thereby more certain of occurrence.

<sup>1</sup> Brennemann J. Abdominal Pain of Throat Infections. *Am Jour Dis Child* 22:493 1922.

<sup>2</sup> Tanaka M and Crowe S J. Direct Blood-stream Infection Through Tonsil. *Arch Otolaryngol* 1:510 1925.

<sup>3</sup> Herrick W W. Extrameningeal Meningococcus Infection. *Arch Int Med* 23:409, 1919.

Since the advent of chemotherapy the probability of these complications occurring has diminished to a great extent.

**Treatment.**—The patient should observe a strict régime of bed rest until the temperature has returned to normal for a day or so.

In an adult the fluid intake should be maintained around 3000 cc per day with a correspondingly lesser amount for younger individuals. The water diuresis thus induced will cause the elimination of significant amounts of bacterial toxins with relief of muscle ache, backache, etc.

Irrigating the pharynx every two hours with 500 cc of a hot (110° to 115° F.) 0.85 per cent saline or a 5 per cent glucose solution will mechanically remove bacteria from the mucous membrane as well as induce a beneficial local hyperemia.

Full doses of penicillin or sulfadiazine should be given and maintained until the temperature has returned to normal. The local use of one of the powdered sulfonamides or suspensions or penicillin solution insufflated or sprayed into the nose and throat three or four times daily seems to be very beneficial.

### CHRONIC PHARYNGITIS; GRANULAR PHARYNGITIS, LACUNAR PHARYNGITIS, CLERGYMAN'S SORE THROAT

This disease is usually characterized by symptoms, such as irritability and dryness of the throat.

**Etiology.**—The chief factors in the etiology of this disease are the irritating effects of infections of the nose, sinuses, tonsils, improper breathing and speaking (public speakers and singers) and possibly smoking.

Many children will have a so-called "compensatory" enlargement of the pharyngeal lymph follicles following a tonsillectomy and adenoidectomy. The exact cause is not known.

Mouth breathers, from any cause, frequently have enlarged lymph follicles of the pharynx. Apparently the dryness of the pharynx associated with persistent mouth breathing has something to do with the hyperplasia.

Exposure to excessive dust and irritating gases is a probable factor in many instances.

Certain general systemic diseases such as the various types of rheumatic disorders, hepatic cirrhosis, and cardiac affections may have a predisposing influence upon the chronic type of pharyngitis. Metabolic disturbances, dietary errors and endocrine dyscrasias may also be factors in certain people.

Heredity is a factor in the lymphoid hyperplasia in many instances.

**Pathology.**—The changes in the mucous membrane consist at first of an increased hyperemia and local leukocytosis, and later of the deposit of the least differentiated cells or connective-tissue cells. That is, hyperplasia of the mucous membrane occurs. The lymph tissues around the tubular glands of the pharynx are hyperplastic, enlarged and raised above the surface of the mucous membrane. Occasionally

the tubular glands in the center of the lymphoid masses are filled with a whitish exudate or cheesy material

**Symptoms** Symptoms are frequently absent. The most common symptom when present is a sensation of dryness or soreness. There may be a frequent desire to clear the throat or even a dry hacking cough.

In aggravated cases the voice becomes hoarse after moderate use especially in public speakers though the cords may be neither red nor inflamed. The hoarseness may be due to a spasm of the muscles of the pharynx and irritation of the superior laryngeal nerve.

The secretions in the early stage of the disease are excessive, thick and tenacious. At a later stage the glandular functions become impaired and the throat dry and glazed.

Upon examination of the pharynx the mucous membrane appears redder than normal at least in certain areas. In other areas it is pale

and fibrous in appearance especially in old chronic cases. Enlarged blood vessels often extend across the posterior pharyngeal wall. The secretion is often thick, heavy and mucopurulent though in the later stages it may be scanty and only forms a film over the surface. In these cases the patient complains of dryness of the throat. The uvula is often relaxed and elongated.

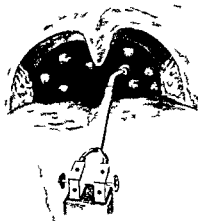


FIG. 164. Showing the cautery point applied to pharyngeal follicular glands in the treatment of follicular pharyngitis. From four to five follicles may be thus treated at a sitting under cocaine anesthesia.

The lymph follicles of the posterior wall and of the lateral walls behind the posterior pillars of the fauces are enlarged. This condition is often referred to as *pharyngitis hyperplastica lateralis*, a needless subdivision of chronic pharyngitis. The follicles are sparsely distributed on

the posterior wall of the pharynx but are closely grouped along the lateral walls. They appear as yellowish red raised areas on the posterior wall and as nodular elongated masses behind the posterior faucial pillars.

**Treatment**—In the early congestive stage simple astringent and demulcent local remedies combined with the regular use of a mild aperient mineral water may be of help. In the more advanced cases in which hyperplasia of the mucous membrane has occurred and in which the lymph follicles are hyperplastic improvement will only follow the destruction of the tubular glands around which the lymph masses are located.

Local caustics such as silver nitrate and chromic acid may reduce the enlarged lymph follicles

In well advanced cases the lymphatic nodules whether discrete or massed as they may be on the lateral walls behind the posterior pillars of the fauces should be punctured with a cherry red cautery electrode (Fig 164) The mucous membrane should be brushed once or twice with a 10 per cent solution of cocaine and from four to five hyperplastic follicles burned out with the electrode At the end of the fifth or sixth day four or five more follicles may be treated in a similar manner and so on until they are all destroyed This course of treatment is often very beneficial though it may fail if the infected tonsils are not removed or a nasal infection cleared up

Instead of using the cautery roentgen ray radiations ultra violet light or diathermic treatment of the follicles may be used with good results

The internal administration of iodine such as potassium iodide Lugol's solution etc is of distinct help in most instances

### AGRANULOCYTOSIS

**Synonyms** — Agranulocytic angina malignant neutropenia

Werner Schultz<sup>1</sup> is given credit for first describing the syndrome called agranulocytosis characterized by an abrupt onset of malaise prostration high temperature and usually an ulcerative and gangrenous angina of the mouth and throat accompanied by a severe leukopenia with a marked reduction in the granulocytes Senator in 1888 described a similar condition as did Brown<sup>2</sup> in 1902

**Etiology** — It seems well established that amidopyrine dinitrophenol and possibly other closely related drugs are etiologic agents in agranulocytosis It is known that derivatives of the benzene ring with their known toxicity to the bone-marrow are capable of producing a depression of the neutrophiles The possibility has been advanced that the disease might be due to an idiosyncrasy hypersensitivity or allergy of these drugs The highest incidence of the disease has been reported in countries which use the largest amounts of the organic compounds aniline dyes and coal tar drugs It is not limited to any one class or occupation

Several cases of agranulocytic angina allegedly due to the sulfonamides salicylates arsenic thioricil and the gold salts have been reported in the literature

The disease is more prevalent in middle-aged women but the incidence is without limitations as to sex and age

A specific organism capable of producing the disease has not been found The bacteria most frequently recovered in the clinical cases are streptococci and staphylococci

<sup>1</sup> Deutsch med Wchnschr 48 1495 1922

<sup>2</sup> München med Wchnschr 35 47 1888

<sup>3</sup> Am Med 3 649 (April) 1902

In many of the cases there is a history of some form of oral surgery in the presence of Vincent's angina just prior to the attack.

Focal infection has been considered but attempts to prove it as an etiologic factor have been unsuccessful.

It has been thought to be due to a deficiency of purine and nucleotide production in the body.

Heredity is supposed to have no influence.

Other possible etiologic factors that have been mentioned are embryonic deficiency of the bone-marrow, the use of prophylactic typhoid serum and diphtheria antitoxin, roentgen ray, menstruation and pregnancy but definite evidence has not been presented in regard to any of them.

**Pathology — Throat Lesions —** The lesions in the mouth and elsewhere vary from a small superficial ulcer to a widespread gangrene. The location of the first lesion of the pharynx and mouth is as a rule on the

surface of the tonsil, the middle of the anterior pillar and the gum margins. The early throat lesion is a slight injection producing a scratchy feeling. The throat soon becomes diffusely congested and bright red in appearance. A membrane is usually present either in the region of the tonsils or along the gingival margins extending at times to the uvula and pharynx. Somewhat later necrotic ulcers appear in these regions with very little inflammatory reaction. They are covered with a dirty gray or dark necrotic membrane (Fig 16a).



FIG 16a. Agranulocytosis. Ulcers are present on the tonsils, anterior pillar, uvula and gums.

Microscopic examination of the ulcers shows a central area of necrosis surrounded by a zone of lymphocytes and leukocytes with an absence of granulocytes. Cultures of the lesions usually show the staphylococcus, streptococcus and Vincent's organisms.

Other ulcerative mucous membrane lesions are found at times in the colon, rectum, vagina, urinary bladder, trachea and bronchi.

The submaxillary lymph nodes are much enlarged as a rule.

**Granulocytopenia —** Agranulocytosis is characterized by the destruction or paralysis of the granulopoietic tissues, particularly of the bone-marrow. It is expressed by a marked diminution or absence in the number of the leukocytes circulating in the blood. The essential change seems to be a hypoplasia of the myelopoietic tissues. The diminution in the number of granulocytes may be due to a destruction of the cells or a failure of development.

A profusion of young granulocytic cells may be found; however, the mature cells are greatly reduced in number. The blood changes may antedate the clinical manifestations.

The blood picture as a rule shows the total leukocyte count as less than 2000 cells per cubic millimeter. In the fulminating form this may fall as low as 200 in which the granulocytes may be absent. A terminal rise in the number of immature granulocytes has been noted (Krumbhaar).

The mononuclear leukocytes as a rule show little change. The erythrocytes fail to show any change except in the chronic form in which a secondary anemia is present.

The blood cultures are positive in from 10 to 15 per cent of the cases. The organisms recovered are the pneumococcus, streptococcus hemolyticus and viridans, *Bacillus coli communis* and *B. pyocyaneus*.

The change in the bone marrow is usually an aplasia characterized by the disappearance of the granulocytes and their precursors; however the bone-marrow may show a myeloid hyperplasia. Areas of necrosis are seen. In both forms the granulopoietic cells show marked retrograde changes.

**Symptoms.** Marked prostration is the most common symptom. It comes on early in acute cases, is usually pronounced during the entire course and varies somewhat in proportion to the granulocytic content of the blood.

The history reveals that the patient is easily fatigued, short of breath and irritable. A slight fever is present at some time during the day as a rule. A history of frequent attacks of sore throats or infection of the oral cavity is usually obtained. As the disease progresses the elevation in temperature increases with chilly sensations or even a severe chill at times.

Bleeding often occurs both as petechial hemorrhages and from the ulcerative lesions.

The symptoms and blood changes are usually present for some time before the local lesions of the mouth and throat are observed.

**Diagnosis.**—The diagnosis is made from the presence of severe prostration, fever and depression associated with lesions of the mouth or elsewhere and a leukopenia with an agranulocytosis characterized by an absence of myelocytes with normal red blood cells and platelets.

**Differential Diagnosis.**—The differential diagnosis should be made from aleukemic leukemia, monocytic angina, aplastic anemia and leukopenias of certain metastatic bone tumors, arsenic, gold and benzol poisoning and various septic processes.

The absence of anemia with normal hemoglobin and blood platelets should distinguish agranulocytic angina from the first three conditions except in the final stages of aleukemic leukemia when an anemia may be present.

Monocytic angina has a more favorable prognosis. The disease lasts from three to four weeks. The entire lymphatic system is swollen. The blood shows about 80 per cent large mononuclear cells.

The lesions in the mouth and throat may be confused with acute leukemia, infectious mononucleosis, Hodgkin's disease, acute follicular tonsillitis, Vincent's angina and diphtheria.



**Prognosis** — The prognosis is extremely grave. The mortality rate has been between 80 and 90 per cent, however, recent reports give a much more favorable prognosis.

The disease seems to be becoming quite rare, probably due to the diminished consumption of aminopyrine both alone and in various combinations.

**Treatment** — The benzol derivatives should be avoided, especially mixtures of amidopyrin and the barbiturates.

Adenine sulfate, a split product of the pentose nucleotides, given intravenously in a dosage of from 1 to 2 gm. or more daily has given the greatest success. One gram of adenine sulfate dissolved in 35 cc. of saline by boiling may be given intravenously every eight hours for about ten doses.

Vitamin B<sub>6</sub> (pyridoxine hydrochloride) given in 200 mg. intravenous doses daily seems to increase the formation of granulocytes by stimulating the formation of myelocytes in the bone-marrow. Penicillin given in adequate doses controls any concurrent infection.

The roentgen rays and liver extract have been used for stimulating the blood-forming organs with some success.

Repeated blood transfusions seem to be of great aid.

### LEUKEMIA

The cause of leukemia is unknown. It may occur at any age.

Leukemic involvement of the mucous membrane of the nose may occur as a hemorrhage or as a leukemic infiltration. The hemorrhagic form is the most frequent. Nosebleed is usually the first symptom of the nasal involvement and is often overlooked until other symptoms of the disease appear. It occurs in cases of chronic as well as in those of acute leukemia. The cause for the bleeding is not known. In every case of persistent and severe nosebleed a complete blood count should be made. Hemorrhages in the skin of the nose occur usually as petechiae. Leukemic infiltration of the skin of the nose may occur in many forms.

The most frequent symptom of leukemia involving the mouth is bleeding gums. The gums may be pale and of normal contour or hypertrophic and edematous. Areas of necrosis may appear on the cheek lips, gums or palate. Vincent's angina should be differentiated.

The most frequent lesion of the throat is the marked enlargement of the tonsils accompanied by much pain. A peritonsillar abscess may be suspected. Necrotic lesions of the tonsil or pharynx occur frequently. The tonsil may be entirely destroyed. This condition should be differentiated from tonsillitis, diphtheria, tuberculosis, syphilis and Vincent's angina.

The leukemic involvement of the larynx usually takes the form of areas of necrosis. These may be small, shallow areas involving all or a part of the laryngeal structures. Petechial hemorrhages may occur the same as in the oral or pharyngeal cavities. Hemorrhagic blebs are more rare. The necrotic lesions of the larynx must be differentiated from laryngeal diphtheria, Vincent's angina, syphilis, carcinoma and tuberculosis.

Cervical adenopathy is a frequent finding in both the acute and chronic leukemia.

The areas of necrosis are probably due to Vincent organisms which invade the mucous glands and lymphatic structures gaining a foothold with subsequent breaking down of the tissue. The spirochetes are thought to be the causative organism in the destruction of tissue (Love). The treatment is symptomatic. The disease is fatal.

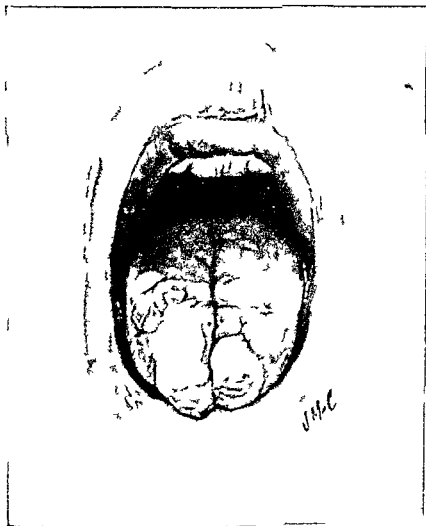


Fig. 166. Leukoplakia of the tongue.

### LEUKOPLAKIA

Leukoplakia of the mouth and throat is a chronic painless keratinization of the mucous membrane. The milky white patches are usually found on one or more regions of the tongue, palate, buccal mucous membrane, posterior gums, floor of the mouth, the angles of the mouth, and at times in the larynx.

**Etiology** — The exact cause is unknown. It is thought to be due to long continued irritations such as that from tobacco or ill fitting dentures. Chemical or mechanical irritations of any type may be factors in the etiology. It is most common in men past middle age.

**Pathology** — The keratinization is the result of a proliferation of the superficial layer of the mucous membrane and sometimes of the filiform papillae. It may occur as a smooth area, a raised plaque or a papillomatous type. It is considered a precursor of cancer in some instances.

**Symptoms** — The onset is insidious and without symptoms. In a later stage there may be sensations of burning or irritation. Palpation usually gives an indurated feeling to the examining finger.

The differential diagnosis should be made from syphilis, lichen planus, burns and various ulcerations. A biopsy may be necessary to differentiate.

**Treatment** — Any etiologic factors should be eliminated or corrected. The teeth and mouth should be kept clean. Applications of irritating substances such as silver nitrate should be avoided. Carbon dioxide snow, radium plaques, electrocoagulations, the galvanocautery and surgical excision have all been used with some success.

## PEMPHIGUS

Pemphigus (Fig. 167) is a slowly progressive disease of the skin and mucous membranes characterized by the formation of bullae by remissions and usually by death.

**Etiology** — The cause of pemphigus is not definitely known. It has been attributed to the various strains of streptococci, the bacterium pemphigi of Ibersson, and endocrine or trophic disturbances.

It usually occurs in adult life with a slight prevalence of females over males. A high percentage of the cases are in Russian Jews.

**Pathology** — Four types may be recognized: (1) Pemphigus acutus, (2) pemphigus chronicus or vulgaris, (3) pemphigus foliaceus and (4) pemphigus vegetans.

Pemphigus acutus usually runs a rapidly fatal course. A few cases recover. Pemphigus chronicus is the type most commonly encountered on the mucous membrane.

Pemphigus foliaceus is characterized by exfoliation with involvement of the mucous membranes at a later stage.



FIG. 167 — Pemphigus of the pharynx. Note the fibrinous exudate over the palate and pharynx and the two bullae on the left soft palate and anterior pillar (New Arch Otolaryngol.)

*Pemphigus vegetans* is rare. The lesions are elevated, becoming papillomatous in appearance.

A benign type of pemphigus may last for years

**Symptoms.**—The patients usually complain of a sore throat or mouth, difficulty in swallowing and salivation

The first lesions on the mucous membranes of the mouth pharynx and larynx are seen as small blisters which later rupture. These blisters sooner or later coalesce and include large areas of the mucous membrane. Due to secondary infection a foul odor is present. Ulceration and a fibrinous exudate may be present in severe cases.

**Differential Diagnosis.**—This must be made from Vincent's angina, syphilis, tuberculosis, sprue, erythema multiforme, ulcerative and aphthous stomatitis, and at times leukoplakia

**Treatment.**—The treatment of this condition is very unsatisfactory. Davis and Davis report some success with coagulen and iron cacodylate. New and O'Leary report no improvement in their cases with this treatment. Arsenic is at present the drug in greatest favor. Supportive treatment with concentrated liquids and soft foods is indicated.

Lever and Talbott<sup>1</sup> report good results in the treatment of pemphigus vulgaris with adrenal cortex extract, dihydrotachysterol or massive doses of vitamin D.

### THRUSH. OIDIOMYCOSIS

Thrush is a stomatitis caused by species of *Monilia*, as well as varieties of *oidium*. It is characterized by the presence of small, pearly-white flecks and patches, usually multiple and scattered over the mucous membrane of the mouth, predilecting such sites as the lateral margins of the tongue, inner surface of the cheeks, palate and fauces. The lesions suggest and resemble somewhat the deposit of coagulated milk, but are adherent, and when forcibly removed give rise to bleeding surfaces of the affected mucosa.

A multiplicity of fungi are capable of producing this disease.

Thrush is preeminently a disease of infants, although it is occasionally found in adults of the debilitated type.

It is more common in the tropics but it is frequently seen in the temperate zones.

The diagnosis is made from the white pearly lesions together with a microscopic examination which reveals the mycelial threads and conidial forms.

**Treatment.**—Ordinary sterilization of the mother's nipples (or rubber nipples) plus cleansing of the mother's and baby's hands should be done.

Various disinfectants such as 10 per cent boroglycerin, 10 per cent sodium hydroxide, 2 to 3 per cent ferric chloride, 1 per cent aqueous solution of gentian violet and 3 to 5 per cent thymol with  $\frac{1}{2}$  to 1 per cent oil of cinnamon prepared in olive oil have been used with successful results in most instances.

Vitamin deficiencies have not been associated definitely with thrush.

<sup>1</sup> New England J. Med., 231, 1, (July 6) 1944

However intensive therapy with the entire vitamin B complex or other vitamins may be indicated

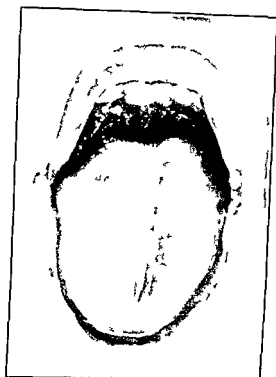


FIG. 165.—Lichen planus of the tongue and buccal mucosa. Diagnosis of oral lichen planus may be difficult if the characteristic lesions are absent

### LICHEN PLANUS

Lichen planus in the mouth may consist of gray or dull white dots with a stellate or a delicate lacework arrangement

The etiology is unknown. Dental diseases, poor fitting plates and smoking may be factors

The symptoms may be absent. A rough feeling or a burning sensation may be mentioned

The treatment consists of the removal of all mouth irritants and infections. The general body conditions should be treated as indicated. Roberts<sup>1</sup> recommends deep intramuscular injections of bismuth twice a week for two or three months

There seems to be a marked tendency to recurrence

### GRANULOMATOUS ULCER OF THE NOSE AND FACE

Idiopathic granulomatous ulcers of the nose and face have been reported by various authors under various titles. Hoover reviews the literature and reports 4 cases

<sup>1</sup> Ann. Otol. Rhinol. and Laryngol. 42: 385 (June) 1933

<sup>2</sup> Arch. Otolaryngol. 34: 865 (November) 1941

**Etiology.**—The cause is unknown. The more common diseases associated with ulceration of the nose and face such as syphilis, tuberculosis, leprosy, glanders, fungous infections, new growths etc., must be excluded as an etiologic factor.

The disease is more common in males than females (20 to 1) and is usually observed in young adults or middle-aged persons.

**Pathology.**—The pathologic picture is that of an amorphous granulating mass with ulceration accompanied with some inflammatory reaction but with no characteristic findings.

**Symptoms.**—The onset is very insidious covering a period of months or years before ulceration develops. Symptoms may be absent during the pre-ulcerous stage other than a nasal congestion beginning nasal obstruction or a watery discharge. Headaches may be mentioned in some instances.

As the granulomatous tumor develops the nasal obstruction increases and is not relieved by shrinking solutions. A serosanguinous or purulent nasal discharge develops usually with a disagreeable odor. Crusts may form and when removed leave an ulcerative surface with slight bleeding.

In the late stage ulcers appear on the external surface exposing necrosis of the bony structure of the nose. An abscess related to the lacrimal sac may form and drain (Hoover). Other abscesses may form on the palate or alveoli with loss of teeth.

A mild or even a septic type temperature with chills may develop during the active ulcerative stage. If an artery is eroded active severe bleeding may take place. The course of the disease varies from six months to two or three years.

**Prognosis.**—The prognosis is usually hopeless but a few apparently spontaneous cures have been reported.

**Treatment.**—The treatment of these granulomatous ulcers has been very unsatisfactory. Surgical excision and removal by electrocoagulation has given temporary relief only. Roentgenotherapy has given some good results in the early stage but seems to have no value in the more advanced stages. The sulfonamides and/or penicillin may be used locally and internally to control the secondary infection.

### BENIGN SARCOID

Multiple benign sarcoidosis of Boeck, a disease of the reticulo-endothelial tissues, is characterized by the formation of hard granulomas or tubercles usually of the skin and occasionally of the upper respiratory tract although any organ of the body may be affected (Poe<sup>1</sup>).

**Etiology.**—The disease is thought to be a form of tuberculosis although tubercle bacilli are rarely found in the lesion. Young individuals are most often affected.

**Pathology.**—The hard tuberculoid, non-inflammatory growth contains accumulations of epithelioid cells usually surrounded by a thin layer of lymphocytes. Giant cells of the Langhans type are scattered

<sup>1</sup> Ann Otol, Rhinol and Laryngol, 51: 430 (June) 1942

throughout. Caseation and necrosis are absent as is ulceration of the surface epithelium. The histologic picture resembles that of tuberculosis. The growths may vary in size from that of a pin-head to a large plaque. They have been reported as occurring as one or more lesions in various portions of the larynx, mouth and nose.

**Symptoms.**—Symptoms, if present, would be those of any smooth, hard, benign growth depending upon the size and location. The disease is chronic and may last for years.

The diagnosis is made from the appearance of the lesion and from the histologic examination. The tuberculin reaction is usually negative.

**Treatment.**—The treatment of benign sarcoid is very unsatisfactory. The lesions have been treated by electric desiccation but without complete success. Fletcher<sup>1</sup> removed sarcoid nodules from the turbinate and ethmoid regions in 2 cases by surgical excision without recurrence.

### NOMA

Noma (cancerum oris, gangrenous stomatitis) is "a rapidly progressing gangrene, beginning on the gums or mucous membrane of the cheek and leading to extensive sloughing and destruction" (Tribble and Dick<sup>2</sup>).

The etiology is unknown. It is usually seen between two and ten years of age in poorly nourished children who live in unsanitary environments, or who are in debilitated health. No specific organism has been found as the causative agent. It has been attributed to Vincent's organisms.

The prognosis is very unfavorable as there is no response to any known treatment. The mortality rate has been given as from 75 to 90 per cent.

The differential diagnosis should be made from agranulocytic angina, leishmaniasis, syphilis, jaws and gangosa.

### GANGOSA

**Synonyms.**—Rhinopharyngitis mutilans, granuloma gangrenosa, kaninoma.

Gangosa is described by Myerson<sup>3</sup> as "a destructive condition of the palate, nose, and certain exposed skin surfaces of the body."

**Etiology.**—The etiology and method of spread are not known.

The disease is largely confined to the Pacific Islands, but has been observed in certain parts of Italy or in large seaport towns. Some observers report that a majority of the cases occur in women, others believe that males and females are equally affected. It has been reported in all ages except infancy. It is especially prone to occur in natives who live in unsanitary conditions.

Propinquity may be an influencing factor in the spread of the disease. It has been attributed to a specific organism such as a type of tertiary jaws or a form of syphilis. Most observers consider it a distinct entity.

<sup>1</sup> Arch Otolaryngol, 39, 470, (June) 1914

<sup>2</sup> Arch Otolaryngol, 16, 11 (July) 1932

<sup>3</sup> Laryngoscope 43, 394, (May) 1933

**Pathology.**—"The disease begins as a small nodule which rapidly becomes an ulcer, usually in the mid-line of the hard palate, but it may begin on the mucosa of the soft palate or the posterior pharyngeal wall" (Myerson). The ulcerative process may destroy the hard and soft palate, nose, eye and sinuses. In mild cases a perforation of the septum, or an ulceration of the soft palate and posterior pharyngeal wall may be the extent of the destruction.

The disease may last a few months to many years. Most cases ultimately recover. The general health remains good as a rule.

### INFECTIOUS MONONUCLEOSIS

Infectious mononucleosis (acute glandular fever) seems to be a distinct clinical entity. It occurs in epidemics or sporadically. It is characterized by: a marked enlargement and tenderness of the cervical lymph nodes; fever lasting from one to three or four weeks, an acute throat infection; and an enlarged spleen.

Examination of the blood shows an initial leukopenia of from 3000 to 5000 white blood cells followed by a leukocytosis of from 10,000 to 30,000 in the great majority of the cases. The count may reach 40,000 or even 60,000 in some cases (children). The leukocytosis is due to an increase of the lymphocytes, many of which are atypical but not immature. The red blood cells, hemoglobin and platelets are usually normal. A positive heterophil antibody serological test (sheep agglutination) confirms the diagnosis.

There are various clinical types observed such as the acute angina form with high temperature, the abdominal and the insidious or chronic type.

The insidious type may occur with little or almost no temperature but with a well marked lymph node enlargement.

The acute anginal type occurs most commonly in children. Suppuration rarely happens. The disorder improves spontaneously as a rule. Complications are rare. The continued lymphocytosis as well as the lymph node and splenic enlargement may persist for weeks or even months in some instances.

The mortality is low (1 to 2 per cent).

The disease should be differentiated from German measles and other acute infectious diseases of the upper respiratory tract.

### ORAL AND PHARYNGEAL LESIONS ASSOCIATED WITH DERMATOLOGIC CONDITIONS

Oral and pharyngeal lesions associated with dermatologic conditions are varied. They embrace such common conditions as syphilis, tuberculosis, lupus, leukoplakia, neoplasms, the exanthemata, Vincent's angina, and canker sores. Other rare conditions as mentioned by Montgomery<sup>1</sup> are blastomycosis, coccidioidal granuloma (endemic in

<sup>1</sup> Ann Otol, Rhinol and Laryngol, 46, 179, (March) 1937.



certain regions in California), torulosis (due to the *Cryptococcus hominis* which usually attacks the brain and meninges), sporotrichosis (usually in the form of ulcers), actinomycosis, tularemic ulcers, xanthoma scleroma (Mikulicz or foam cells containing Irish's encapsulated bacillus associated with plasma cells and Russell bodies), lymphoblastomas (Mycosis fungoides, Hodgkin's disease, leukemias and lymphosarcoma) fungi (hairy tongue or black tongue, usually actinomycosis), perleche (due to *cryptococcus* or *Monilia*, usually occurring in children as a smooth, grayish-white thickening on the mucous side of the commissures of the mouth), thrush (moniliasis), lichen planus, vitamin deficiencies (pellagra and scurvy), acrodynia, allergic gingivitis and stomatitis and erythema multiforme.

Certain drugs may produce eruptions of the mucosa as well as the skin. These drugs are phenolphthalein, barbiturates, quinin and arsphenamine.

Stomatitis, ulcerations and bullæ may occur from antipyrine, acetanilide, salicylates, bromids, iodids, barbiturates, quinin, cinchophen, etc.

Pigmentation, gingivitis and stomatitis may result from the heavy metals such as bismuth, mercury, lead, gold and silver. Pigmentation of the buccal mucosa may occur also in Addison's disease, acanthosis nigricans and hemochromatosis.

The mucous membrane may be involved in "scleroderma, herpes zoster, impetigo, granulomatous conditions such as yaws, leprosy, leishmaniasis, lymphopathia venereum, granuloma pyogenicum, moluscum contagiosum and occupational dermatoses" (Montgomery).

Any acute infectious process of the nose or throat should receive proper attention. The application of the rays of light and heat from an infra-red lamp to the neck at the angle of the lower jaw may be helpful.

### ELONGATED UVULA

The cause of elongated uvula is not known other than a possible hereditary influence. It has been attributed to a chronic nasopharyngitis.

The uvula may be slender and pendulous, or it may be enlarged and pendulous.

**Symptoms**—In most subjects it causes but slight symptoms or none at all. In sensitive patients it often causes a reflex cough when it touches the epiglottis or the base of the tongue. The cough may be spasmodic and is usually dry. Nausea and vomiting especially early in the morning are sometimes mentioned.

**Treatment**—In mild cases astringent lozenges afford relief. The uvula may also be painted with astringent solutions of alum, tannic acid or with epinephrine. In the more severe cases amputation is indicated. In all cases the nasopharynx and the oropharynx should be examined and any diseased conditions treated.

**Surgical Treatment**—The uvula should be painted with a 10 per cent solution of cocaine. The tip of the uvula is then seized with forceps and drawn directly forward. While in this position the tip should be severed.

with heavy blunt scissors, as shown in Figure 169. By cutting the uvula in front while drawn anteriorly, the bevelled cut surface of the stump faces posteriorly. This is a point of practical importance, as in swallowing solid food the raw surface is not irritated by it.

**Casselberry's Operation**—Secure anesthesia by puncturing the uvula with a 10 per cent solution of cocaine.

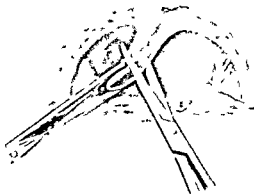


FIG. 169.—The amputation of the elongated tip of the uvula just below the lower extremity of the muscle. The scissors are so applied that the posterior surface of the uvula will be the wounded surface. This lessens the irritation in swallowing food and in breathing through the mouth.

Seize the tip of the uvula with forceps and draw it directly forward. While in this position an upward and medianward cut is made with scissors to the central axis of the uvula. A similar cut is made on the opposite side, thus removing a wedge-shaped piece of the uvula, as shown in Figure 171.

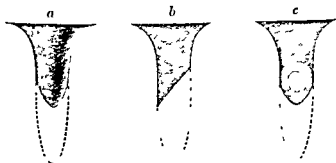


FIG. 170.—Three views of the amputated uvula. *a*, anterior view, *b* lateral view, *c*, posterior view.

The anterior and posterior cut edges of the wound are then secured with two or three black silk sutures, black thread being used, because it is easier to see at the time of its removal. Yankauer's needles may be used with advantage. The sutures should be removed at the end of three days.

Hemorrhage has been reported after uvulotomy. This may be avoided by limiting the amputation to the portion of the uvula below its muscular fibers—that is, only the thin relaxed portion should be removed, as the blood vessels of the uvula do not extend beyond the muscular fibers.

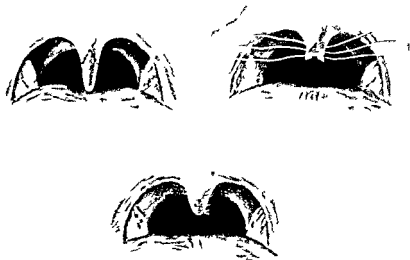


FIG. 111. Casselberry's operation for elongated uvula.

### CALCULI OF THE SALIVARY GLANDS

The presence of calculi in the salivary glands or their ducts is more common than is apparent if one considers only the reported cases. Harrison<sup>1</sup> found 375 cases in a review of the literature from 1825 to 1926. To these he adds 27 cases of his own. They occur most frequently in men in middle life.

Calculi may form in all of the salivary glands or their ducts, but the larger proportion—about 60 to 70 per cent—are found in the submaxillary gland or duct. The parotid and sublingual glands each furnish from 15 to 20 per cent. They are found more frequently in the ducts than in the glands.

Salivary calculi are composed largely of phosphate and carbonate of calcium. The lesser constituents are calcium fluoride, sodium and calcium chloride, and a small amount of sulphocyanide of potassium. Animal matter and bacteria form a small portion. The salts of the saliva are deposited in the duct or gland.

The causes are thought to be (1) inflammation of the mucous membrane with an increased precipitation of salts, (2) the deposition of salts around bacterial masses or inorganic particles.

<sup>1</sup> Surg. Gynec. and Obst. 40:431, 1926.

The calculi give symptoms when they grow sufficiently large to block the duct or from infection induced by stasis of the gland and duct. When the duct becomes blocked the affected gland becomes swollen and tender more especially after eating. Irtial dryness of the mouth is a common complaint.

Frequently the calculus can be felt by palpation or a probe may be inserted into the duct producing a rough grating sensation when the tip of the probe comes into contact with the calculus. Pressure on the swollen gland may force the saliva or pus around the obstruction and out of the lumen of the duct into the mouth.

The roentgen ray shows the presence of calculi in from 70 to 80 per cent of the cases.

**Wharton's Duct** — The duct of Wharton after leaving the submaxillary gland runs forward and upward beneath the mucous membrane of the floor of the mouth over the mylohyoid muscle. It lies between the lingual nerve above and the hypoglossal below until the surface of the genioglossal muscle is reached where the lingual nerve crosses the inner side. The duct opens on the floor of the mouth through a papilla beside the frenum of the tongue. The duct is from  $\frac{1}{4}$  to  $\frac{1}{2}$  cm. in length.

Probing the duct may be dangerous as an acute exacerbation of the infection with a secondary cellulitis may result.

**Differential Diagnosis** — The differential diagnosis of the submaxillary swelling should be made from lymphadenitis in the submaxillary area, acute dental abscess with swelling in the submaxillary triangle, carcinoma of the submaxillary gland, Ludwig's angina, ranula, mixed tumors of the submaxillary gland with swelling in the submaxillary triangle, syphilis and tuberculosis.

Swelling of the parotid salivary gland due to calculus must be differentiated from benign tumors, carcinomas and sarcomas, swellings of the preauricular lymph nodes due to pyogenic infection, syphilis, mumps, tumors of the submaxillary and sublingual glands, ranulas and mucous cysts of the floor of the mouth, lymphadenitis due to infection in the teeth or tonsils, and Mikulicz's disease.

**Treatment** — Occasionally a calculus may pass spontaneously. The use of belladonna to inhibit the flow of the secretion of the gland is contraindicated.

Calculus in the duct is removed under local anesthesia as a rule. The stone is fixed either by clamping the tissue below and behind the stone with forceps or a suture. An incision is made in the long axis of the duct and the stone removed with tissue forceps or a scoop curette.

Extirpation of the gland may be indicated if multiple stones occur within the gland associated with a chronic inflammation, chronic strictures of Wharton's duct, or frequent recurrence of the calculus.

If the calculus occurs in the sublingual duct an incision over the calculus is usually sufficient for its removal.

Care should be taken to avoid the sublingual artery, lingual vein and lingual nerve which are located between the duct and tongue. In remov-

ing calculi from the submaxillary gland the close proximity of the lingual and hypoglossal nerves should be remembered

### MIKULICZ'S DISEASE PSEUDOHYPERTROPHY

Mikulicz's disease is applied to cases in which the salivary and lacrimal glands show symmetrical swellings. Other glands may be involved. The pathogenesis is not known. Infections may play a part. A generalized disease of the glandular system may be a factor. The glandular tissue is replaced with lymphatic cells.

The treatment is symptomatic. Medication with iodine, arsenic and roentgenotherapy have been recommended.

### PLUMMER-VINSON SYNDROME

The Plummer-Vinson syndrome is characterized by anemia, dysphagia, glossitis and achlorhydria.

**Etiology**—The exact cause of the syndrome has not been determined. It is nearly always found in women and is frequently associated with achlorhydria and occasionally with a splenic tumor. The achlorhydric anemia is not necessarily related to the syndrome. It has not been determined whether the anemia precedes or is the result of the dysphagia.

**Pathology**—The anemia is that of an essential hypochromia. The glossitis takes the form of an atrophy of the lingual papillæ. The smooth red tongue appears shrunken. Fissures at the corners of the mouth (cheilitis) and spoon nails are frequently observed. Various lesions in the upper portion of the esophagus have been reported such as cracks, abrasions, hyperkeratosis, adhesions, webs, bands, etc. The hyperkeratinization of the epithelial lining of the esophagus may be pre-cancerous.

**Symptoms**—The dysphagia usually expressed as a tightness or cramping of the throat and referred to the level of the larynx, seems to be the outstanding symptom. The dysphagia may be of sudden onset and subsequent attacks separated by long intervals or the pain may be of gradually increasing severity. The dysphagia has been attributed to a hysterical type of spasm or to lesions of the mouth of the esophagus such as webs or bands or any lesion which may interfere with the sensory arc of the swallowing reflex. The patient frequently complains of a pain in the atrophic tongue.

Symptoms and signs associated with the anemia, cheilitis, spoon nails and splenomegaly would be found when these conditions are present.

**Treatment**—The treatment should be directed to any etiologic or pathologic conditions found.

### ABSCESS OF THE PAROTID GLAND

**Etiology**—An inflammatory process (other than mumps) within the parotid gland with or without an abscess formation may be due to a blocking of Stenson's duct from inflammation, foreign objects especially

salivary calculi, a direct extension of an infection from neighboring structures especially the fascial spaces of the neck or as a complication of abdominal or pelvic operations. Furstenberg<sup>1</sup> stresses the importance of dehydration as the most important etiologic factor in the latter condition.

**Symptoms.**—The symptoms of an abscess of the parotid gland are pain, tenderness and swelling of the gland especially after eating. Symptoms and signs of various degrees of sepsis are present as from any abscess formation.

**Treatment.**—The palliative treatment is adequate hydration, the sulfonamides and/or penicillin as indicated, hot moist external dressings, suberythreal doses of roentgen rays to the affected part, large doses of the compound solution of iodine and gentle probing of Stenson's duct.

If definite evidence of suppuration is present and the above measures have failed the abscess must be incised and drained.

**Technic.**—An incision is begun at the level of the zygoma and extended downward along the ramus of the jaw to the angle, then forward 2 or 3 cm along the inferior margin. The skin and subcutaneous tissues are reflected forward exposing the lateral surface of the parotid gland. Numerous incisions are made into the compartments of the gland as necessary for adequate drainage. Large drainage tubes are inserted and left in place for one or two weeks or until suppuration has ceased. Irrigations with solutions of penicillin may be made through the tubes.

<sup>1</sup> Jour. Am. Med. Assn., 117: 1594 (November 8) 1941.

## CHAPTER XIX

### DISEASES OF THE TONSILS

INFECTIONS of the tonsil may be classified according to their anatomic changes as.

1. Inflammatory in which the changes may be acute congestive, acute suppurative or chronic ulcerative or gangrenous

2. Hyperplastic in which the hyperplasia may be of the physiologic type (hereditary, endocrine glands, etc) or due to chronic inflammation

3 Atrophic either physiologic or as the result of chronic inflammation.

Various combinations of the above anatomic changes may be observed in the same tonsil at the same or different times

In inflammatory processes focal cellular infiltrations are found localized in areas of the epithelium. The epithelium may be absent or eroded in spots. If the epithelium is eroded the superficial portion of the lymphoid tissue will show this cellular infiltration as well. Granulation tissue is also formed in these eroded areas. A normal or thickened squamous epithelium may cover these areas later. Active ulcers, with polymorphonuclear infiltration of the base are occasionally present. The purulent exudate found in the tonsillar crypts is characteristic of acute rather than chronic infections.

The hyperplastic tonsils following chronic inflammation contain more fibrous tissue than the physiologic type. Both have giant follicles with large germinal centers. Narrow borders of closely packed lymphocytes are found around the follicles. Lymphocytes and plasma cells may be seen between the epithelial cells. The epithelium may show vacuoles.

A normal or physiologic atrophy of the tonsil is shown by the absence of leukocytes, lymphoblasts, mitoses and the diminished size and number of the follicles. The apparent increase in the fibrous tissue is due to the relative loss of lymphoid tissue. The tonsillar crypts approach the fetal type, that is they become flat and shallow. Epithelial cysts, surrounded with scar tissue, may be seen. Bone and cartilage occur in from 10 to 35 per cent. They may be regressive inflammatory manifestations rather than congenital.

#### ACUTE CONGESTIVE TONSILLITIS

Acute congestive tonsillitis is almost always associated with a generalized nasopharyngitis. As this subject has been discussed under pharyngitis and rhinitis further consideration will not be given here.

#### ACUTE LACUNAR TONSILLITIS

**Synonyms.**—Acute follicular tonsillitis, acute suppurative tonsillitis, cryptic tonsillitis

**Etiology**—The various general etiologic factors discussed under Acute Inflammatory Diseases of the Nose Throat and Sinuses would apply to acute tonsillitis and will not be reviewed here. The chief local causes of this and other forms of tonsillitis are the local impairment of the epithelium of the crypts and the invasion of certain pathogenic bacteria.

**Local Lesion of the Tonsil**—Bacteria are only absorbed through dead or impaired cryptic epithelium. Hence the prime requisite for tonsillar infection is an impairment of the cryptal epithelium. This condition may be brought about by the retention of exfoliated epithelium and other debris in the crypts of the tonsil. The retention is formed by the constriction of the mouths of crypts from previous inflammation and by the plica triangularis and plica semilunaris which cover the mouths of some of the crypts in such a manner as to prevent the expulsion of their contents. The toxin thrown out by the imprisoned microorganisms causes the death of the cryptal epithelium and thus opens the way for the invasion of the microorganisms into the tonsil and the general lymphatic and circulatory systems, hence the constitutional symptom in this disease.

**Bacteriology** (See Acute Hemolytic Streptococcic Throat Infections, page 240)—The bacteriology of acute tonsillitis embraces several pathogenic microorganisms. By far the most frequent organism found in the acute form is the hemolytic streptococcus and the *Streptococcus viridans*. The pneumococci and staphylococci are found less frequently.

Tonsillitis is frequently associated with the specific fevers such as scarlet fever, measles, diphtheria, etc.

Most investigations show that the organisms in many cases persist and reinfect the individual or spread to others. It would seem that a large percentage of the adult population of the class seeking private consultation present definite evidences of tonsillar disease associated with the presence of pathogenic organisms producing under conditions favorable to general infection more or less serious systemic symptoms. In the chronically ill typical healthy tonsils are the exception rather than the rule.

The disease is most common in young adults between the twentieth and thirtieth years of life. It is also common in children and more rare after the fortieth year of life.

**Pathology**—In acute lacunar tonsillitis the tonsil is swollen though the chief changes occur in the crypts where there is an accumulation of leukocytes and dead epithelial cells intermixed with pathogenic bacteria. The transudation of leukocytes occurs chiefly through the cryptic membrane rather than the surface mucosa. The accumulated material in the crypts or lacunæ is sometimes entangled in a fibrous exudate or pseudomembrane though the pseudomembrane is not always present.

**Symptoms**—In this as in other acute infectious processes the onset is sudden and is attended by malaise and fever. Chilly sensations or light rigors may mark the attack. The temperature gradually rises until the end of the first to the third day to 102° or 103° F. and in young



children it may rise as high as  $104^{\circ}$  to  $105^{\circ}$  F. The febrile movement is accompanied by soreness upon swallowing which as the disease progresses may become quite painful. The inflammation extends to the pharyngeal mucous membrane and even in exceptional cases to the eustachian tube and the middle ear. There may be pain in the ear through reflex sources without actual inflammation in the tympanum. Tinnitus and slight deafness may also be present. The lymph node under the angle of the jaw is usually swollen and tender as it is in a state of great physiologic activity in its attempt to check the invading host of bacteria. The swollen condition of the tonsil, cervical lymph nodes and surrounding muscles renders rotary motions of the head somewhat painful. The same condition also renders articulation and phonation imperfect the voice being thick and indistinct. The tongue is

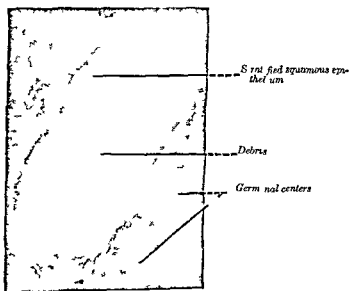


FIG. 172.—Tonsil crypt filled with debris ( $\times 80$ )

coated with a yellowish brown fur and the breath is fetid and offensive. Transient albuminuria is sometimes present especially if the attack is severe and prolonged.

The acute symptoms rarely extend beyond the fifth or seventh day. The febrile movement and the swelling and soreness rapidly subside until the temperature is normal and the act of deglutition and the rotation of the head may be performed with comfort to the patient. The patient though convalescent is often left in a very weakened condition.

Examination of the tonsils during the early acute stage shows them to be red and swollen with the crypt openings covered with yellow spots of exudate (Plate IV A).

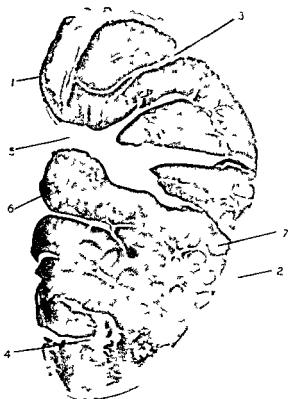
The patches are not true membranous products as found in pseudo-

# PLATE IV



## Acute Lacunar Tonsillitis

The tonsil is red and swollen. The crypt openings are covered by a yellow exudate.



## Section of a Tonsil Removed on Account of Chronic Lacunar Disease

- 1 Supratonsillar margin 2 Fibrous capsule of tonsil 3 Trabeculae or septa
- 4 Degenerated and mechanically lacerated crypt 5 Dilated tonsillar crypt
- 6 Epithelial surface 7 Lymphoid tissue

membranous and diphtheritic inflammations but are the secretions and debris which completely fill the crypts. Occasionally a fibrinous exudate is admixed with the debris which gives it some of the characteristics of an inflammatory membrane. The protruding secretion and debris are easily wiped away in contradistinction to the diphtheritic membrane which is closely adherent to the epithelium.

The adenoid if present and the lingual tonsil are usually simultaneously inflamed with the faucial tonsil and the yellowish exudate or debris peculiar to the faucial tonsil is usually found in the shallow clefts of the adenoid and still more shallow depressions of the lingual tonsil. The debris is similar in composition to that found in the crypts of the faucial tonsils.

**Diagnosis** — Acute lacunar tonsillitis should be differentiated from diphtheria. The following table will aid in the differential diagnosis although there are cases in which the differential points are obscure and dependence must be placed upon the bacteriologic findings.

<i>Acute lacunar tonsillitis</i>	<i>Diphtheria</i>
1 Onset marked by sharp rise of temperature	1 Onset rise more gradual
2 Rapid bounding pulse	2 Slow feeble pulse
3 Depression not marked	3 Depression marked
4 Exudation limited to the tonsil especially the mouths of the crypts	4 Exudation extends beyond the tonsils and is not limited to the crypts
5 Exudate not adherent	5 Exudate closely adherent
6 Exudate soft and friable	6 Exudate firm and leathery
7 Exudate not distinctly membranous	7 Exudate membranous and may be removed in strips
8 Swollen lymph nodes uncommon except in severe cases	8 Swollen lymph nodes common even in mild cases
9 Albuminuria occasionally present	9 Albuminuria common
10 Klebs-Loeffler bacillus absent	10 Klebs-Loeffler bacillus present

**Treatment** If temperature is present the sulfonamides and/or penicillin should be given as indicated.

This type of tonsillitis is more amenable to local treatment than any other. One application of a 25 per cent solution of nitrate of silver if applied locally during the first twenty four hours of the disease in many instances will abort the attack. Subsequent applications may be given as necessary. In applying silver to the tonsil the excess of fluid should be squeezed from the cotton wound applicator to prevent it trickling to the larynx where it will produce violent spasm of the intrinsic muscles. The silver salts are not well tolerated by the motor nerves and muscles of the larynx and severe suffocative symptoms may be produced by inattention to the technic of its application.

The local insufflation of one of the powdered sulfonamides directly onto the tonsils and into the crypts seems to give excellent results. The powder may be applied two or three times daily. The throat and tonsils may be sprayed four or five times daily with a 5 to 20 per cent suspension of sulfathiazole microcrystals in place of the powder if desired. The value of the sulfonamides impregnated into chewing gum lozenges etc. in treating tonsillitis is questionable as the active ingredient may not reach the tonsils in sufficient quantity to be effective.

The exact evaluation of penicillin sprays (250 to 500 units per cc) has not been determined at the present time

Suction through a glass tonsil suction tube (Hurd) will help in evacuation of the crypts

If there is a history of repeated attacks of acute lacunar tonsillitis the tonsils should be removed during the interval between the attacks

### HYPERPLASIA OF THE TONSIL

**Etiology**—Hyperplasia of the tonsil usually begins early in life and continues until young adulthood when it usually ceases active development and often undergoes an atrophic change. Repeated attacks of acute tonsillitis or a long continued chronic inflammation of the tonsils may result in a hyperplastic condition of the lymphatic cellular structure

A hereditary tendency to overgrowth of the lymphatic structures of the body including the tonsils is present in many instances. Occasionally the hyperplasia is present from birth and may continue through life

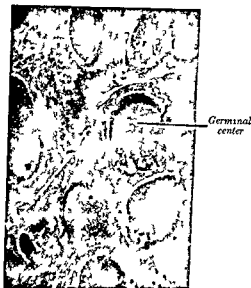


FIG. 173.—Hyperplasia of the tonsil in a child (X 30). Microscopically the hyperplastic tonsil of a child presents a picture of great cellular activity with many large germinal centers where mitoses are abundant. There is little fibrous tissue. In an adult hyperplasia of the tonsil does not present such cellular activity. Germinal centers may be numerous but they are small and mitoses are not as numerous. There is an increase in fibrous tissue.

**Pathology**—In hyperplastic tonsillitis the enlargement is due to an increase in all the cellular structures composing the tonsil whereas in a fibrotic tonsil the connective tissue cells are relatively increased to the other cellular elements. The hyperplastic tonsil in children shows great cellular activity with many mitoses in the numerous germinal centers. In a child the tonsil is soft and smooth in outline whereas in an adult it is often much harder and is nodular in outline. In children

the tonsil is so loosely attached to the sinus tonsillaris that it can be removed easily without force with its capsule intact. In many adults the tonsil is loosely attached though it is ordinarily more firmly attached than in children.

The hyperplastic tonsils may have healthy crypts but as a rule the reverse is true. The lining epithelium of some of the crypts is usually of low vitality, often hornified and is unable to resist the invasion of pathogenic microorganisms.

**Symptoms**—The symptoms of hyperplastic tonsillitis are usually those of obstruction to breathing especially in children. If a moderate hyperplasia only is present this obstruction may be absent. Frequent sore throats and colds may be complained of or occasional earaches or middle-ear infections. As a rule there is an accompanying hyperplasia of the other lymphoid structures of the throat and of the lymph nodes as well.

**Treatment**—Palliative treatment directed toward the removal of the caseous plugs from the crypts and from the pocket formed by the union of the plica triangularis with the tonsil may be instituted when for any reason an operation cannot be performed.

If an hypertrophied or hyperplastic tonsil gives rise to untoward local symptoms, to constitutional disturbances or to local lesions in remote portions of the body it should be removed.

**Röntgen Ray Treatment of Tonsils**—Many patients with hyperplastic tonsils are seemingly benefited for a period following roentgen ray treatments. However hyperplastic tonsils harboring the usual infectious material if controlled bacteriologically are found not to be affected by the roentgen ray. The much shrunken tonsil which is the end result claimed for irradiation is often found clinically to be as great a menace as is the enlarged tonsil. A possible field for roentgen ray treatment may be found in reducing the lymphoid hyperplasias on the posterior wall of the pharynx persisting after tonsillectomy.

### CHRONIC FIBROTIC TONSILLITIS

**Definition**—Chronic fibrotic tonsillitis is a disease of adults as a rule. It may be a physiologic atrophy of the tonsil without symptoms but is frequently characterized by the more or less continued presence of pus in the crypts. Accompanying this pus in many cases are masses of caseous material composed of layers of desquamated epithelial cells enclosing cholesterol crystals, fatty matter, leukocytes, microorganisms and occasionally calcareous deposits. The masses vary in size from that of a grain of wheat to that of a small bean. The crypts most often involved are those which open into the supratonsillar fossa and those covered by the plica semilunaris (tonsillaris).

This latter type of fibrotic tonsillitis frequently acts as a focus of infection causing various pathologic lesions in remote portions of the body.

**Bacteriology**—The organisms found in the fibrotic tonsils are the usual pathogenic varieties such as the *Streptococcus viridans*, *Strepto-*

coccus hemolyticus Staphylococcus pneumococcus and several varieties of Gram negative diplococci. The Streptococcus viridans Streptococcus hemolyticus and a Gram negative diplococcus are most frequently encountered.

**Symptoms**—Local symptoms may be absent or if present are not usually severe in character. The patient may complain of a sticking pain upon swallowing. Some patients have the sensation lasting perhaps for only a minute or two of a foreign body lodged in the tonsil.

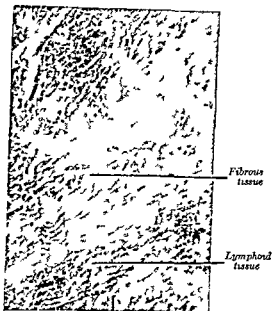


FIG. 174.—Fibrosis and atrophy of the tonsil ( $\times 80$ ). There is a decrease in the size of the germinal centers which are few and scattered with a much lessened cellular activity. A great increase in fibrous tissue also is present and a thick trabecula can be seen.

The patient frequently coughs up the caseous masses which have a fetid odor. Upon exerting pressure upon the tonsil with a flat instrument the pus and caseous masses are forced from the crypts especially from the superior ones.

The repeated removal of the plugs affords some relief and their tendency to reform is diminished though a cure by this procedure does not often occur.

The caseous masses in themselves are not necessarily indicative of a great amount of absorption of toxins; however, where these masses are present, fluid pus can usually be demonstrated by pressing on the tonsil. This fluid pus is very significant of a chronic absorption of bacteria or toxins and in the presence of some condition due to a focus of infection the advisability of removing the tonsils should be seriously considered.

These infected fibrotic tonsils are subject to acute exacerbations generally of a mild type, the mucous membrane becoming slightly

reddened. There is also some soreness upon swallowing. The temperature is but little elevated and may attract no attention.

Occasionally the mouth of a crypt becomes closed by inflammatory adhesions (caseous encyst) and the yellowish color shows through the thin membranous covering over the mouth of the crypt.

**Complications**—Infections of the tonsils are frequently the cause of such systemic conditions as infectious arthritis, acute rheumatic fever, nephritis, endocarditis, myocarditis, anemia, cervical adenitis, goiter, chronic coughs and cold, and a large group of nervous disorders commonly classified as neurasthenia. This is discussed under the subject of The Tonsils as Loci of Infection.

**Treatment**—If the symptoms annoy the patient and recur at frequent intervals, or if the patient has evidence of infection in a remote part of the body which may reasonably be assigned to absorption through the tonsils, they should be removed.

If the patient refuses removal or removal is not advisable for other reasons, local irrigations of the crypts with a saline solution, suction applied to the crypts, and local antiseptic applications will give some relief.

## VINCENT'S ANGINA

**Synonyms**—Ulcerative tonsillitis, pseudomembranous angina, trench mouth.

**Etiology**—Since Vincent described a spirillum associated with a fusiform bacillus found in certain forms of ulcerative tonsillitis and gingivitis, the condition has been called Vincent's angina. The disease is most frequently found in young persons, though it occurs often in those of middle and later life. A debilitated state of health, local irritative lesions in the mouth, such as decayed teeth, inflamed gums, and oral uncleanness, favor the development of the disease, which is by no means an uncommon one. A low vitamin C content of the blood seems to be an important factor in the etiology in many individuals. There is a widespread geographic distribution of the disease, but it is more common in the temperate and tropical climates than in the arctic regions. It is present under endemic conditions in the Americas, France, and the Orient. Epidemics have been reported. It is frequently spread from one person to another from kissing, towels, dishes, etc.

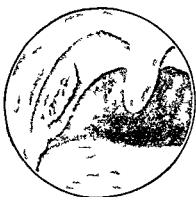


FIG. 17a—Vincent's angina of the tonsil.

**Pathology**—The lesions commonly involve one tonsil, usually at its upper part, may spread to the soft palate, the other tonsil, the pharynx, or the gums. It may even spread to the larynx, trachea, and ears. The

membrane covering the patches is a pseudomembrane and is formed by the necrosis of the superficial layers of the mucous membrane not by exudation. The patches are of a grayish white color surrounded by a red inflamed areola but separated from each other by healthy tissue. On removal of the pseudomembrane which is granular and cheesy in consistency an ulcerative area is exposed varying in extent and depth. The ulcerated areas bleed freely and are soon covered by a new pseudomembrane. The ulceration at times is very destructive destroying the whole or a portion of the tonsil and invades healthy tissue. The spirillum and the fusiform bacillus penetrate the tonsil substance to a considerable depth. They are more numerous in the immediate vicinity of the ulcer.

The microscopic examination of a fresh smear taken directly from the ulcer or a section of the pseudomembrane stained with Loeffler's methyl blue or fuchsin show fusiform bacilli twice as long as wide pointed at the ends and with this a spirillum forming a network around the bacilli. The spirillum is 10 to 20 microns in length. This being the only fusiform bacillus occurring in the mouth is readily recognized when found associated with the spirillum. These bacteria grow best on an acid medium. They will live but not multiply under aerobic conditions. Lunnichiff has shown that the spirochete precedes the fusiform bacilli in the invasion of the tissues.

**Symptoms** — The usual symptoms are a subacute or mild tonsillitis sore mouth and gums headache general malaise chilly sensations temperature varying from normal to 102.5° F. There may be no constitutional disturbances the patient complaining only of more or less pain on swallowing tender or bleeding gums or he may have discovered the yellowish patch on examining his throat or gums with a mirror because of a slight feeling of discomfort. Occasionally the symptoms are most violent great pain on swallowing or talking breath fetid more or less gastric disturbance submaxillary and cervical lymph nodes enlarged and tender.

The ulcer may be single or the membrane may spread like diphtheria and is rapidly. As a rule the ulcers are sluggish grayish brown in color and bleed easily.

An early sign in children is a painless unilateral enlargement of the cervical lymph nodes.

This disease is acute subacute and often becomes chronic the ulcers persisting for weeks or months. One attack is likely to be followed months or even a few years later by a recurrence. The organisms frequently occur without symptoms.

**Diagnosis** — The diagnosis is made by the discovery of the typical Vincent's bacteria the fusiform bacillus and spirillum in a smear. Unquestionably many cases occurring in both children and adults suspected of being diphtheria but in whom Loeffler's bacillus is not found are cases of Vincent's angina. Such suspected cases in whom the culture is negative for diphtheria should be examined by means of a smear for Vincent's angina. The same may be said of doubtful cases of suspected syphilis both in the secondary and tertiary stage. An examination of a smear would clear up the diagnosis.



**Differential Diagnosis**—The diseases usually confused with it are diphtheria and syphilis. Many cases of what are called ulcerative sore throat, gangrenous tonsillitis, are in reality Vincent's angina. Noma and acute leukemia should be excluded.

**Prognosis**—While most attacks are more or less mild, the patient suffering only local discomfort, the disease tends to persist for several days or weeks, and recurrence may occur at any time. Complications are seldom troublesome, and a fatal issue is not to be expected unless the larynx or trachea becomes invaded.

The fusiform bacilli and spirochetes seem to be much less profuse in their growth when associated with the diphtheria bacilli than when associated with the streptococci. They become more active in the presence of pyorrhea, decayed teeth or tonsils that are chronically infected. In severe infections of the gums, teeth are frequently lost, and the destruction of the gum tissue and alveolar process around and between the teeth establishes food pockets, which sooner or later result in the further loss of teeth.

**Treatment**—The disease responds rapidly to the parenteral administration of penicillin. Frequently 20,000 units given every three hours for five doses is sufficient.

Topical applications of a penicillin solution in a concentration of 500 units per cc. four or five times daily is a rapid and effective therapeutic measure in most instances. Glossitis and stomatitis have been reported (Phillips<sup>1</sup>) from the use of penicillin lozenges and troches in a few cases.

Sulfathiazole tablets (0.5 gm.) dissolved on the tongue every two hours for two or three days give rapid relief of symptoms and seems to be much more effective than the older methods of treatment.

A 10 to 50 per cent solution of nitrate of silver is an excellent astringent as is a 5 per cent chromic acid, trichloroacetic acid or a 10 per cent solution of copper sulfate. The strong silver nitrate solutions shrink the gums and tonsillar tissue, permitting better aeration and access to the hidden areas. A 30 grain to 1 ounce solution of zinc sulfate is also recommended.

The sodium perborate treatment of Vincent's gives excellent results. A thick paste of the chemically pure salt should be made with water and spread over the affected areas. The patient should hold the paste in the mouth for about five minutes. A solution should be used as a gargle three or four times a day. The sodium perborate splits up in the mouth forming nascent oxygen.

Arsphenamin or other forms of arsenic have been used extensively in recent years on the theory that it is a specific for the spirochetes. It may be dissolved in glycerin and used locally or given in the vein as in syphilis. Sodium cacodylate 1 to 3 grains given intramuscularly or subcutaneously is very satisfactory.

Bismuth and sodium tartrate in 1:5 per cent aqueous solution given intramuscularly and the glycerinated solution applied locally in the same strength has given good results in many cases.

In all cases oral and dental cleanliness should be maintained.

Adequate amounts of citrus fruits or of vitamin C should be prescribed. Nicotinic acid 100 to 200 mg daily seems to be beneficial in most instances.

To prevent infecting others drinking and eating utensils should be sterilized and kept separate. Sputum and mouth discharges should be burned. Following the attack local disease of the mouth and teeth should be attended to but operative work should be postponed until making sure by the microscope of the absence of the specific bacteria.

### HYPERKERATOSIS OF THE TONSIL MYCOSIS LEPTOTHRICA

Hyperkeratosis of the tonsillar tissue is characterized by the appearance of numerous white projections not only from the cryptal orifices of the tonsils proper but also from the orifices of the lymph follicles on the posterior and lateral pharyngeal walls and on the lateral glosso-

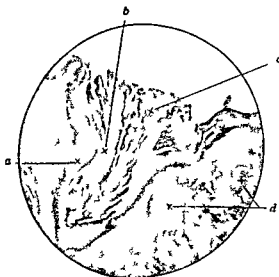


FIG 1 C—Hyperkeratosis showing the typical appearance under low power. The horny mass is growing from a comparatively small area of the cryptal epithelium and the plug shows the ordinary fraying of its edges. *a* cryptal epithelium *b* horny mass *c* masses of bacteria *d* follicles (Wood)

epiglottidean folds. This condition does not occur on portions of the throat where there is no lymphoid tissue. The lymphoid tissue of the upper respiratory tract however is so ubiquitous that occasionally we may see the little white projections on almost any part of the mucosa. In the large majority of cases the condition is limited to the faucial and lingual tonsils. That it reaches its greatest development on the base of the tongue and at a position just behind the lateral glosso-epiglottidean folds and the posterior part of the inferior poles of the tonsils is due almost entirely to mechanical reasons. The contractions of the muscles during swallowing prevent food from coming in intimate contact with

the surface of these parts and therefore permit the projections to grow undisturbed. Although the horny material is quite resistant to trauma the bacterial accumulations which form the greater mass of the projection are easily brushed off so that the size of the growth is much greater where it is protected from mechanical disturbances.

Hyperkeratosis is a condition peculiar to young adults and is self limited, from two to three years being required for it to run its course.

**Symptoms**—The symptoms caused by this condition of the throat are either entirely wanting or very slight and are due for the greater part to the local irritation caused by the hard horny plugs. If they project from the base of the tongue so as to come in contact with the epiglottis there is an irritating tickling sensation which causes a hacking cough. If they are so placed as to be compressed during the act of swallowing they may give rise to a slight pricking pain.

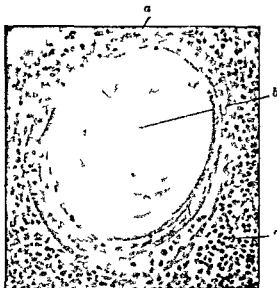


FIG. 1. Hyperkeratosis. Cross-section of the terminal portion of a crypt showing the concentric arrangement of the layers of horny material and the epithelium which is still somewhat integrated. *a* epithelium *b* horny material in crypts *c* lymphoid tissue (Wood).

Occasionally among the rich and various bacterial flora which grow in such luxuriance on this horny material there may lurk a germ possessed of more or less pathogenic power which may set up an accompanying inflammatory reaction in the tonsil or surrounding structures.

**Pathology** Wood states. In hyperkeratosis the epithelium loses its rarefied condition and becomes ordinary pavement squamous epithelium similar to that covering the surfaces of the tonsil except that generally it does not show the connective-tissue papillæ. The crypt of the tonsil is markedly dilated and filled with a horny mass which merges at various points into the epithelium.

According to the mechanical circumstances by which the tonsil is surrounded the horny mass becomes sooner or later broken up into

lavers between which multiply and grow organisms of all varieties. This fraying of the cryptal plug may take place within the crypt itself so that the resulting fissures permit the bacteria at times to penetrate almost but not quite to the living epithelium.

The toxins elaborated by these organisms must be absorbed to a greater or less extent by the tonsillar tissue. It is probably due to the fact that the cryptal epithelium has become an intact protective barrier that a more noticeable reaction is not a common result.

**Treatment**—Treatment is unnecessary though if the horny masses cause irritation they may be removed by cauterization. The electrocautery should be used to destroy them and the surrounding tissues should be penetrated until only healthy tissue remains. From three to four masses may be treated at each sitting at intervals of one week.

### CALCULUS OF THE TONSIL TONSILLOLITHS

Small quantities of calcareous or gritty particles are often found in the center of the caseous plugs filling the crypts of the tonsil in chronic lacunar tonsillitis. They sometimes become quite large and fill the crypts and are known as calculi of the tonsil or tonsilloliths. They occur much more frequently in adults than in children and at times they reach an extraordinary size. Such patients usually give a history of repeated attacks of tonsillitis in earlier years.

Ihrenfried<sup>1</sup> believes fungi to be the essential factor in the etiology of tonsilloliths. He finds the tonsilloliths to be composed largely of calcium phosphate and bases their association with fungi on the affinity of the latter for lime salts.

The calculi are rounded or oval as a rule but with a somewhat roughened granular surface. They are usually yellow or yellowish gray. A concentrically laminated or radially striated structure may be indistinctly shown in many cases.

C. V. Weller made a careful research of the sections from a series of 1000 consecutive pairs of tonsils. It was found that 80 showed calcareous deposits in the crypts of one or both tonsils an incidence of 8 per cent. The microscopic study did not indicate any special age of incidence in respect to the genesis of the smaller calculi.

Among the group of 80 positive cases of microscopic tonsillar concretions of the crypts 47 or about 59 per cent were developing in the large so-called actinomyces-like colonies of mixed mouth organisms so commonly found in the crypts of the tonsil. This is the most common mode of origin. Such colonies grow peripherally with a more or less clearly rayed or clubbed outer zone. As they become of large size the central portion dies and in the dead center lime salts may be laid down. In 33 or 41 per cent of the 80 positive cases the origin of the tonsillar calculi was found to be in the accumulated keratohyalin masses in the crypts.

In the masses of keratohyalin so commonly found in the crypts of the tonsil calcification begins in certain of the older hyalin laminae so that

such an area when seen in microscopic section shows elongated striæ of lime salt indicating the roughly concentric layers of the original material

In the adenoid calculus formation occurs especially in the mucopurulent exudate and in keratohyalin

**Symptoms**—The symptoms are identical with those of chronic lacunar tonsillitis with caseous plugs in the crypts, that is there are recurrent attacks of mild tonsillitis with redness which is especially marked around the affected crypts. The patient may be aware of a constant sensation as of a foreign body in the throat. The breath is often fetid.

**Treatment**—The treatment consists in the removal of the tonsils or in the removal of the calculus as in chronic lacunar tonsillitis. If the calculus is not easily disengaged from the crypt an incision of the wall of the crypt facilitates its removal. Pain may be obviated by injecting a 2 per cent solution of procaine into the substance of the tonsil in the region of the calculus.

### CARTILAGE AND BONE IN THE TONSILS

Isolated portions of bone and cartilage found in the tonsils are embryonic rests of the branchial arches in many instances. They are found only in the connective-tissue structures of the tonsil.

According to Weller cartilage is formed by a process of progressive transformation (direct metaplasia) of the connective tissue while bone in the tonsil always arises in such newly-formed cartilage by processes wholly comparable to endochondral ossification elsewhere.

Weller found the incidence of cartilage and bone to be 20.9 per cent. Hillekowitz reports of 750 tonsils examined, 54 revealed the presence of cartilage, a percentage of 7.2. Wilkinson found cartilage and bone in 11.21 per cent of 10,000 pairs of tonsils examined.

The occurrence of bone in the tonsil is frequently caused by an abnormally long styloid process extending into the tonsil.

Symptoms are absent as a rule.

### TONSILLAR ABSCESS (PHLEGMONOUS TONSILLITIS)

Phlegmonous tonsillitis is an abscess within the substance of the tonsil. It is not so common as peritonsillar abscess.

One of the tonsillar crypts usually becomes closed thereby creating a closed abscess. Injury to the tonsil may produce the condition.

The *symptoms* are similar to peritonsillar abscess except they are not so severe.

The *treatment* is incision and drainage of the abscess with the subsequent removal of the tonsils.

### PERITONSILLAR ABSCESS (QUINSY)

Peritonsillar abscess is an acute abscess in the peritonsillar tissue.

**Etiology**—The cause is about the same as that given under acute lacunar tonsillitis. Peritonsillitis (quinsy) probably results from an

infection of the crypts in the supratonsillar fossa which are large slit like cavities with irregular outlines and which are in intimate relationship with the posterior and outer aspect of the tonsil. The disease is common in young adults and rare in children.

**Pathology**—In the majority of cases of peritonsillar abscess the pus finds its way into the supratonsillar fossa which is characterized by marked swelling and edema of the soft palate to the extent that the tonsil is pushed downward and mesially.

In another class of cases the pus burrows downward and backward displacing the tonsil forward with little if any swelling in the supratonsillar region. The posterior pillar bulges to a marked extent with abscess in this location.



FIG. 178.—Peritonsillar abscess. The left anterior pillar and palate are distended. The tonsil is pushed to the center and the uvula to the opposite side.

If the superior constrictor muscle is penetrated by the abscess an infection in the parapharyngeal space results. This penetration may take place directly or through the veins. The pus may burrow downward from this locality and enter the mediastinum. The great vessels of the neck may become implicated in rare instances. It is probable that cases terminating fatally belong in this group.

**Symptoms**—The onset of the peritonsillitis is continuous from a preceding acute lacunar tonsillitis as a rule. The temperature rarely exceeds  $99^{\circ}$  to  $100^{\circ}$  F. whereas in the early stage of an acute tonsillitis it often rises to  $103^{\circ}$  F. or higher.

The pain progressively increases with the extension of the purulent accumulation until it is almost unbearable. The muscles of mastication are encroached upon by the abscess hence the patient has the greatest difficulty in opening the mouth sufficiently wide to permit of an examination of the throat. Swallowing becomes difficult and painful. The disease is usually limited to one side. The saliva dribbles from the mouth and forms one of the characteristic symptoms. Lateral movement of the head produces pain on account of the infiltration of the tissues of the neck in the region of the tonsil.

Thick viscid secretion forms in the throat and it is with the greatest difficulty that the patient succeeds in removing it. The tongue is heavily

coated and the breath fetid. Breathing is interfered with on account of the swollen mucous and submucous tissue of the pharynx. The patient has an anxious expression of countenance. During sleep he often has suffocative attacks which awaken him. Laryngeal dyspnea from extension of the edema to the laryngeal tissue is fortunately rare.

Examination reveals a unilateral redness and swelling, as both tonsils are rarely affected at the same time. If both are affected, the second usually begins as the first subsides. If both are affected at once, the suffocative symptoms are more severe and alarming. As the disease progresses, the redness, tenderness, pain, and swelling increase in severity. If the abscess is in the tonsil, the tonsil itself is swollen without much displacement of the palate and uvula. If the abscess is in the peritonsillar tissue, the swelling often appears to be in the region of the upper portion of the anterior pillar. The palate and uvula are pushed over to the opposite side of the throat. This is partly due to the swollen tonsil but largely to the edema, cellulitis and pressure of the abscess itself upon the tonsil. Incisions in this region often fail to reach the pus cavity for this reason, that is, the incision is carried directly into the tonsil instead of into the pus cavity outside of the tonsil.

The soft palate and uvula, as well as the pharyngeal mucous membrane, are red and edematous. The region of the tonsil is a deep, dusky red color. The crypts are often filled with a pulp-like debris, which was probably the original source of infection. The infection does not originate in the peritonsillar tissue but in the supratonsillar crypts of the tonsil as a rule.

Digital examination of the tonsillar region shows more or less distinct fluctuation. The focal center of fluctuation is sometimes located about  $\frac{1}{4}$  inch external to the free border of the anterior pillar, at the junction of the upper third with the middle third of the tonsil, or it may be posterior to the tonsil.

The duration of the disease embraces from five to ten days when allowed to run its course, though it may extend over a longer period. The termination is marked by the spontaneous or artificial discharge of fetid pus. When the discharge is spontaneous it usually takes place through the anterior pillar, though it occasionally occurs through one of the crypts. In some instances there is a spontaneous resorption of the cellulitis without the discharge of pus.

Peritonsillar abscess should be differentiated from infections of the submaxillary space. The latter condition is usually due to caries or infections of the molar teeth. The pus may push the internal muscle against the superior constrictor and hence displace the tonsil toward the median line as in quinsy.

**Complications and Sequelæ.**—Complications and sequelæ are rare. Cases are on record, however, in which the following conditions were present: Edema of the glottis from the downward extension of the process. Strangulation from the spontaneous rupture of the abscess. Thrombophlebitis of one of the large veins of the neck. Ulceration of one of the large arteries in the submaxillary region. Chronic peritonsil-

litis with an intermittent flow of pus. Encysted abscess in the tonsil. Parapharyngeal abscess with or without a mediastinitis.

**Treatment** — Full doses of penicillin or sulfadiazine should be given until the temperature has reached normal and the pain has disappeared.

Hot (110° to 115° F.) 0.5 per cent saline or 5 per cent glucose throat irrigations every two to three hours will hasten recovery and give relief. Heat to the neck and jaw will reduce the muscle spasm from mastication.

**Surgical Treatment** — *Incision* — If fluctuation is present incision and drainage is indicated. The local application of a 10 per cent solution of cocaine or 2 per cent pontocaine solution to the region of the incision is usually sufficient. If marked trismus is present and the patient is unable to open the mouth sufficiently to make the incision applying cocaine through the nose to the descending palatine nerves in the greater palatine canal may give relief from the muscle spasm.

The point of the incision is determined by the location of pouching or fluctuation. It is usually in front of the anterior pillar on a level with the junction of the upper and middle thirds of the tonsil though it may be in the posterior pillar or through the tonsil. Many of the failures to evacuate the pus through the anterior incision are due to the fact that the tonsil often extends forward beneath the anterior pillar. The incision as usually made therefore penetrates the tonsil instead of the tissue outside of it (Fig. 179). If the depth of the incision is carried beyond the outer border of the tonsil, the pus will be found more often. It should be remembered that the anterior third of the tonsil projects forward beneath the anterior pillar, hence in making an incision through the anterior pillar to evacuate the pus it should be made far enough anteriorly to escape the anterior border of the tonsil and should be directed in an outward and a backward direction outside of the capsule of the tonsil. If these anatomic facts are borne in mind the anterior incision will nearly always evacuate the pus if present. If a posterior incision is to be made it should be directed outward through the posterior pillar or in its immediate vicinity, as the pus pocket often extends posteriorly to the tonsil.

**Dissection** — Dissection beneath the anterior pillar to reach the upper pole of the tonsil is seldom necessary but occasionally it may be employed when simple incision fails and pus is thought to be present.

Seize the anterior portion of the tonsil with forceps and pull it medianward and forward to reverse the position of the anterior pillar.

Make an incision at the junction of the anterior pillar and the tonsil thereby partially separating the pillar from the tonsil.

Introduce a blunt dissector through the incision and separate the capsule of the tonsil from the superior constrictor muscle (bed of the sinus tonsillar) until the abscess cavity is reached.

This method of operating can seldom fail to evacuate the pus.

Many writers advocate the complete removal of the tonsil in the presence of peritonsillar abscess especially in cases of sepsis however there are objections to the procedure such as the added risk from the



inhalations of pus if a general anesthetic is used the increased bleeding the increased menace of setting up a systemic infection which occasionally follows the removal of the tonsils when they are the seat of an acute

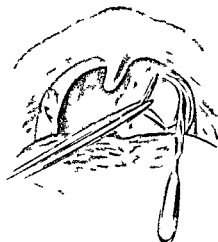


FIG. 19.—The incision back of the capsule of the tonsil to evacuate a peritonsillar abscess. The dissection is started as though the tonsil were to be removed.

infection. Zollner reviewed 91 cases in which tonsillectomy was performed during the acute stage of peritonsillar abscess. In 4 instances in spite of relatively good preoperative conditions fatal septicemia resulted. Tonsillectomy in the acute inflammatory stage may be well borne in general, however there are cases in which it may lead to severe complications.

<sup>1</sup> *Ztschr. f. Hals-, Nasen- u. Ohrenh.* 35: 509 (May 15) 1934.

## CHAPTER XX

### III TONSILS AS FOCI OF INFECTION

THERE is no questioning of the fact that the tonsils are portals of systemic infection. Practically all writers agree that various pathogenic organisms and their toxins gain entrance through the tonsils to the vascular and lymphatic systems the lungs the heart the kidneys and indeed to the whole system through these organs.

Rozenow<sup>1</sup> defines a focus of infection as a place where favorable conditions are afforded for entrance into the blood or lymph stream of bacteria and their toxic products and where they may acquire or maintain peculiar or relatively high invasive power. They make for a forced relationship between parasite and host.

From a practical standpoint only the teeth tonsils sinuses gastrointestinal tract prostate and seminal vesicles and the female generative organs need be considered in searching for a focus. The last three named are relatively infrequent sites of foci of clinical importance in systemic disease.

The tonsils as foci of infection have definitely established themselves as an important factor in a large and growing class of diseases. Judson Diland lists the following systemic diseases having a possible etiologic relationship to chronic infection of the tonsils or sinuses in adults:

*Nervous System*—Meningitis encephalitis bulbar palsy chorea neurasthenia psychoses diseases of the spinal cord cranial and peripheral nerves.

*Bones Joints and Muscles*—Arthritis osteitis periostitis synovitis tendo-vaginitis myositis.

*Alimentary System*—Gastric duodenal and intestinal ulcers gastritis enteritis cholecystitis gall stones cholangitis hepatitis pancreatitis and appendicitis.

*Circulatory System*—Ileocarditis myocarditis and endocarditis hypertension tortitis thrombosis embolism anemia and pernicious anemia.

*Genito-urinary System*—Nephritis pyelonephritis calculus prostatitis seminal vesiculitis endocervicitis and sterility.

*Respiratory System*—Bronchitis bronchiectasis pneumonia bronchopneumonia lung abscess pleuritis and asthma.

*Skin*—Acne furunculosis carbunculosis alopecia herpes herpes zoster urticaria pruritus dermatitis and ichthyosis.

*Eye Ear Nose and Throat*—Conjunctivitis keratitis corneal ulceritis optic neuritis uveal tract disease impaired hearing or deafness tinnitus vertigo and Meniere's disease.

<sup>1</sup> Ann Otol Rhinol and Laryngol 36 4 1927

<sup>2</sup> Ib d 35 4, 1926

The large tonsil with wide-open crypts is not of great pathologic significance so far as acting as a focus of infection is concerned. Of much greater significance is the small buried tonsil flush with the pillars which on pressure yields a fluid pus or shows on examination minute yellowish areas which on incision prove to be tiny abscesses.

Crowe<sup>1</sup> describes the cryptal epithelium and the small ulcers which occur deep in the crypts some of which do not heal but remain chronic. Such ulcers are frequently observed in chronic tonsillitis. Crowe emphasizes the fact that these ulcers are surrounded by a close network of thrombosed capillaries surrounded by a zone of plasma cells, lymphocytes and leukocytes. This condition is encountered most frequently in tonsils removed from subjects suffering from the infectious arthritides. The failure to find any cervical adenitis in some of these patients suggests a direct blood stream infection.

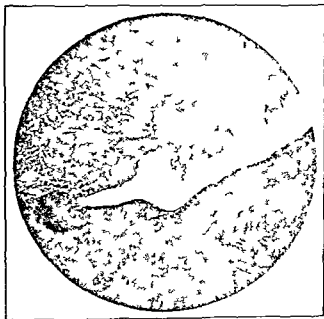


FIG. 180.—Epithelium lining a tonsillar crypt partly destroyed by infection (Evanston Hospital Laboratory)

Rosenow states: It has been proved beyond doubt that in the streptococcal group more perhaps than in any other species of bacteria there are individual strains of almost every degree of virulence. There are strains that affect in diverse manner a wide range of tissues or organs while others have a specific or elective affinity for particular structures without noticeably affecting others.

Mickel<sup>2</sup> has reported on the localizing power of bacteria from foci of

<sup>1</sup> Arch. Int. Med. 33:473, 1924.

<sup>2</sup> Ann. Otol., Rhinol. and Laryngol. 36:4, 1927.

<sup>3</sup> Jour. Am. Med. Assn. 88:1117, 1926.

infection in tonsils teeth prostate and cervix in patients suffering from various diseases. The factor of specificity or elective localizing power was demonstrated in each of six groups of cases studied. He found what Rosenow has observed namely, that the best results in demonstrating this selective action are obtained in cases of acute disease or during periods of exacerbation in cases of chronic disease.

Pemberton<sup>1</sup> reports 400 cases of chronic arthritis observed in army hospitals during the World War. Tonsillar foci of infection were demonstrable in 52 per cent dental in 33 per cent and genito-urinary in 12 per cent. Twenty per cent showed a combination of these.

Holsti<sup>2</sup> in reporting his examination of 203 tonsils from 123 persons found in the arthritic group that a sore throat had recently preceded the attack in 71 per cent of acute cases 47 per cent of relapsing cases of acute arthritis and 22 per cent of chronic cases.

The tonsils and teeth are ideal locations for foci of infection because the areas involved are closed and deprived of oxygen. The lingual tonsils and hyperplastic pharyngeal follicles only occasionally cause systemic disease. When streptococci are confined in the bottom of a crypt closed by adhesive inflammation and deprived of oxygen they gradually acquire virulency pathogenicity and selectivity which acquired characteristics may again be lost in the presence of oxygen. Apparently infected tissue no larger than a small pea is capable of causing a fatal systemic disease. It would seem no relationship exists between the amount of infected tissue and the seriousness of the secondary disease.

Intercurrent infections such as influenza pneumonia etc. or lowered resistance from any cause may convert a harmless into a harmful streptococcus resulting in systemic symptoms.

Infected tonsils may remain a local infection but as a rule even when symptoms are absent toxins or streptococci may enter the blood intermittently or continuously. Tonsils once infected should be considered thereafter a menace and should be removed as a prophylactic measure certainly so in the presence of a systemic manifestation.

A study of the blood by Daland<sup>3</sup> showed that in 40 per cent of cases of systemic disease secondary to tonsillar infection the total number of leukocytes were decreased and the number of lymphocytes increased with a proportional decrease in the polymorphonuclear cells.

Daland states: "In the absence of any other focus the internist suspects chronic infection of the tonsils in an adult when a disease occurs that may be due to that cause as for instance myocarditis cholecystitis arthritis neuritis etc. and this suspicion becomes a strong probability if leukopenia lymphocytosis decreased polymorphonuclear cells usually in association with anemia are also present."

In diagnosing infected tonsils the internist attaches importance to a history of diphtheria scarlet fever quinsy recurring tonsillitis recur

<sup>1</sup> Trans Am Laryngol Assn 33 242 1923

<sup>2</sup> Fenska Lak-Sallak Handl 66 365 1924

<sup>3</sup> Ann Otol Rhinol and Laryngol 35 4 1926

ring sore throat recurring rhinitis enlarged cervical glands a congested or beef like appearance of the anterior pillars the appearance of the tonsils and the presence in pure culture of the *Streptococcus viridans* or hemolyticus in materials obtained from the bottom of a crypt These streptococci when injected into animals may reproduce the disease from which the patient is suffering

Kaiser<sup>1</sup> in studying the results of tonsillectomy found substantial benefits in the incidence of sore throat diphtheria and scarlet fever Cervical adenitis was reduced and early rheumatic attacks were reduced from 30 to 50 per cent in tonsillectomized children

SUMMARY — The tonsils or a tonsil remnant may be suspected as a focus of infection when some disease due to some focus or foci of infection is present and no other focus is found They may also be suspected if there is a history of previous infections of the tonsils with an induration of the tonsil redness of the anterior pillars or expressible pus Enlarged cervical lymph nodes may or may not be present Under these conditions the tonsils should be suspected even though other foci of infection are found

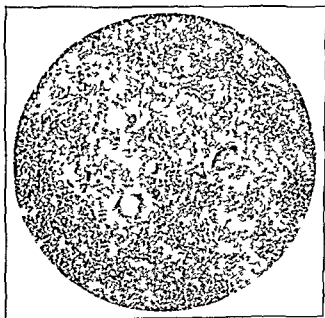


Fig 181 --Tuberculosis of the tonsil Note the extensive fibrosis with necrosis and giant cells (Evanston Hospital Laboratory)

## THE TONSILS AND TUBERCULOSIS

There is still a divergence of opinion as to whether a primary tuberculosis of the tonsils may occur Some hold that the tuberculous process in these glands is direct while others contend that the infection reaches

<sup>1</sup> Jour Am Med Assn 95 837 (September 20) 1930

them from the lungs through the lymphatics and the blood vessels or by the flow of the bronchial secretions over them. Both views are probably correct in selected cases. It is probable however that tuberculous infection of the cervical lymphatic nodes is usually due to the entrance of the bacilli and other microorganisms through the tonsils. This is borne out clinically by the fact that suppurating or tuberculous lymph nodes of the neck are rarely found in phthisical patients. Whereas if they occurred secondarily to pulmonary infection they would be found frequently in such patients.

It would seem from a clinical standpoint that a primary latent tuberculous process may exist in the tonsils or adenoid with no clinical pulmonary signs or symptoms of tuberculosis.

Most observers find an incidence of tuberculosis (giant cells) in tonsils removed at operation at from  $\frac{1}{2}$  to 4 per cent.

## CHAPTER XVI

### TONSILLECTOMY INDICATIONS COMPLICATIONS

**Function of the Tonsils** — The exact function of the tonsils is not known. It is possible that no function exists. Several theories as to a possible function of the tonsils have been suggested: (1) protection against bacterial invasion, (2) glands of internal secretion, (3) a blood-forming organ, (4) an exposed lymph node with a function of elimination or excretion, (5) production of antibodies, and (6) protection of the bronchial tubes from infection.

Much has been written and but little determined concerning the internal secretion of the tonsil. J. Gordon Wilson calls attention to the fact that "The tonsil does not develop like a lymphatic gland from a plexus of preexisting lymph vessels in the mesothelium. It develops as an ingrowth of endothelium from the second branchial pouch and in its origin comes into line with the thymus and the thyroid. The thymus originates from the third branchial pouch, the thyroid from the fourth and the parathyroid from the third and fourth, all by invagination of the endothelial lining of the primitive pharynx."

The only physiologic property the tonsils have been proved to possess is the production of lymphocytes in the germ centers or follicles, and the removal of the tonsils after the first two or three years of life removes only a very small fraction of the normal supply of lymphocytes.

**Indications for Removal of Tonsils** — No set rule can be laid down for the removal of the tonsils, each case requiring special thought. The general indications for tonsillectomy may be listed as follows:

1 Cases of acute suppurative otitis media which have lasted more than six weeks. It is of equal if not greater importance to remove the adenoid as well in this condition.

2 Tonsils which on pressure extrude cheesy matter or pus complicated by symptoms of toxic absorption or a recurrent foul odor of the patient's breath.

3 Infectious material in inflammation of the tonsil may gain entrance to the tube and middle ear either during coughing or vomiting or in intense inflammation by the destruction of the cilia of the epithelium of the tube. Ordinarily the cilia with their wave-like motion carry the secretions from the middle ear to the nasopharynx. When they are destroyed or their action is inhibited by violent inflammation the entrance of foreign matter as bacteria, etc. into the middle ear is comparatively easy. Hence in certain diseases of the ear which have their origin in tonsillar inflammations the removal of the tonsil is indicated.

4 When the cervical lymph nodes are enlarged and tender the tonsils are usually the source of the infection, and if there is a history of repeated or permanent lymph node enlargement the tonsils should be excised.

5 Systemic infection due to a focus where other foci are not found. If other foci are found and fluid pus can be expressed from the tonsils tonsillectomy is indicated.

6 Malnutrition in children in the absence of other causes may be considered an indication.

7 Hyperplasia of the tonsils with obstruction to the breathing is an indication for their removal.

8 Peritonsillar abscess.

9 Diphtheria carriers.

10 Tuberculous infection often begins in the tonsils and when such a process is demonstrated or strongly suspected the tonsils should be enucleated.

11 Unexplained fevers especially in children may come from an obscure tonsil infection.

12 Frequent colds and sore throats are relieved in the majority of cases and may be considered an adequate reason for removal in the absence of other causes. Kaiser<sup>1</sup> found the late results of tonsillectomy seen in 2200 children ten years after operation evident in the reduction of sore throat, cervical adenitis, otitis media, scarlet fever, diphtheria, rheumatic fever and heart disease.

**Contraindications for Tonsillectomy**—Tonsillectomy is usually an elective procedure and the immediate contraindications such as acute local or general infections, syphilis, blood dyscrasias, enlarged thymus, etc., should be corrected before doing the operation. Other contraindications are certain mental and nervous diseases, anomalous blood vessels such as the internal carotid artery and malignant neoplasms of the mouth, pharynx or larynx.

### TONSILLECTOMY

There are many methods of operating upon the tonsils for the cure or relief of the morbid conditions affecting them and the neighboring structures and organs. Only those methods will be described which seem to be the most rational from a clinical and surgical standpoint and which have after long trial given the best results.

**Tonsillectomy with a Tonsil Knife and Snare**—Anesthesia may be either local or general. Local anesthesia is preferable in adults except in those cases in which for various reasons the patient cannot be operated upon in the conscious state. Hemorrhage is usually less under local anesthesia and if bleeding occurs is more readily controlled with the patient awake. The anesthesia is a matter that must be decided by each surgeon as the personal element enters so largely into its consideration. Local anesthesia is contraindicated when operating upon the throats of children, highly nervous or neurotic subjects, in epileptics and status lymphaticus and in some cardiac conditions.

*Local anesthesia* may be induced by a preliminary swabbing of the tonsils and the faucial arches with a 4 per cent solution of cocaine. Following this a 1 or 2 per cent solution of procaine is injected around

<sup>1</sup> Jour. Am. Med. Assn. 95:837 (September 30) 1930.



the capsule (Fig 186) until complete anesthesia is obtained usually 30 to 60 minims to each tonsil is sufficient. 8 to 12 drops of the 1 to 1000 epinephrine should be added to 1 ounce of the procaine solution before injecting.

The position of the patient is a matter of some importance. Under local anesthesia the upright or semi upright position in the operating chair is preferable. The surgeon stands while operating in this position. Under general anesthesia the patient is placed in a supine position upon the operating table. A mouth gag through which ether is administered should be used. The Rose position in which the neck is extended so that the head is in a lower position than the rest of the body will reduce the chances of blood getting into the lungs.



FIG 18 —Street's tonsil hypodermic syringe

In the further description of the technic it is assumed that the patient is conscious and in the upright position.

Seize the tonsil with the vulsellum forceps (Fig 184) the tip of one prong should be placed on the upper portion of the tonsil and the other at the base of the tonsil.

The incision may be started at the upper pole of the tonsil dissecting downward freeing the anterior pillar or the incision may be begun beneath the anterior pillar and carried upward into the supratonsillar space to the posterior pillar. The aim should be to dissect around the upper half of the tonsil forming an inverted U.

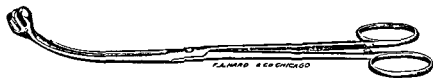


FIG 183 —Ballenger's tonsil forceps

When the upper pole of the tonsil is freed the back of the knife may be used to separate the capsule of the tonsil from its bed unless adhesions are present. If adhesions are present a careful dissection of the adhesions and scar tissue as well as the tonsil is necessary.

The posterior pillar is separated in much the same manner as the anterior pillar. This pillar is not as accessible as the anterior one but it can be brought into view by pulling the tonsil toward the median line and somewhat forward. This puts the attachment of the tonsil to the superior constrictor muscle upon a slight tension turning the

tonsil upon its lateral axis in such manner as to bring the posterior pillar forward and upward where it is readily accessible

The external contour of the tonsil is followed to its inferior portion. At this stage of the operation the use of the knife may be abandoned and a snare substituted to complete the operation. Pass the forceps through the snare and seize the tonsil then pass the snare over the tonsil. Tighten the snare and thus complete the operation.



FIG. 184.—Separating the tonsil from the anterior pillar by means of a curved tonsil knife

If bleeding points persist they should be grasped with a blunt artery forceps and ligated with small sized (No. 0 or 1) plain catgut.

The *plier triangularis* should be grasped with a toothed forceps and removed by means of a wire snare or by means of scissors dissection.

**Tonsillectomy with a Scalpel**—A small scalpel and the vulsellum forceps are required. A tongue depressor is not used as the forceps crosses the tongue and keeps it out of the way.

**Technic**—Induce anesthesia by the injection of the procaine epinephrine solution in the peritonsillar tissue. If the procaine epinephrine solution is used 30 minims may be injected in each tonsil (Fig. 186).

Seize the tonsil with vulsellum forceps one blade on the upper pole and the other at its base as in the preceding method. Pull the tonsil

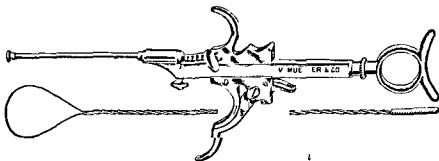


FIG. 185.—Perce-Mueller tonsil snare

medianward and forward to dislodge the anterior shoulder from beneath the anterior pillar. This pulls the posterior margin of the pillar forward and facilitates the introduction of the scalpel between it and the tonsil.

Introduce the blade of the scalpel to a depth of about  $\frac{1}{2}$  inch between the anterior pillar and the tonsil at the junction of the pillar and *plier triangularis* (Fig. 188). Sweep the blade upward to and over the supra

tonsillar margin to the posterior pillar. The knife should be very sharp for this purpose. This completely severs the tonsil from the anterior pillar and exposes the outer aspect of it to further dissection. The upper

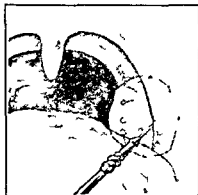


FIG 186 —The various points for injecting the procaine-equinephrine solution around the tonsil. The injections are made between the tonsil and the pillars, not through the pillars or in the tonsil.



FIG 187 —Placing the snare loop over the tonsil after the pillars have been dissected free. The wire loop is tightened thus severing the remaining attachments.

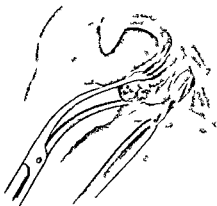


FIG 188 —The first incision in the removal of the tonsil with its capsule intact. The tonsil is drawn forward and medially from the tonsillar fossa. The incision is extended upward over the supratonsillar margin to the posterior pillar.

portion of the tonsil concealed in the supratonsillar fossa is freed from its attachments. If this step of the operation is not observed the dissection is more difficult.

Continue to pull upon the tonsil with the forceps. Then introduce the knife through the upper part of the incision follow closely the capsule of the tonsil and sever it from its attachment to the superior constrictor muscle as shown in Figure 190. The branches of the tonsillar



FIG 189 — Anatomical landmarks of the fauces. *a b* the incision liberating the pillars in the removal of the tonsil. *c* the anterior pillar. *d* anterior pillar. *e* supratonsillar slit-like crypts or hilum of the tonsil. *f* supratonsillar fossa. *g* supratonsillar margin.

artery are severed in this step of the operation. They are small and do not often give rise to severe hemorrhage. If however some of the fibers of the superior constrictor muscle are accidentally removed the main stem of the artery is severed and the hemorrhage may be severe. The bleeding points should be seized and ligated with artery forceps.



FIG 190 The tonsil is separated from the bed of the tonsillar fossa to which it is loosely attached. The capsule is followed closely with the scalpel care being exercised to avoid injuring the superior constrictor muscle which forms the bed of the tonsillar fossa.

The edge of the blade should be slightly turned to the tonsil as this will avoid injuring the superior constrictor muscle of the pharynx.

Disengage the vulsellum forceps from the tonsil and place the tip of one prong in the anterior aspect of the wound the other over the

inner aspect of the tonsil and close them upon the tonsil (Fig 191) Traction the anterior border of the tonsil toward the median line of the throat, using the posterior pillar as a hinge

Then, having rendered the posterior pillar accessible shave it free from the posterior border of the tonsil with the scalpel (Fig 191) Great care should be taken to avoid injuring the muscular tissue of either the anterior or posterior pillars during the dissection

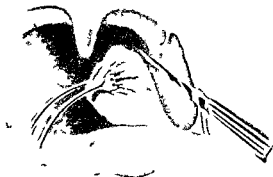


FIG 191 —The tonsil is drawn toward the median line of the throat to expose the posterior pillar to the knife The pillar is incised to the bottom of the tonsil at its junction with the tonsil

The tonsil is now only attached at its inferior portion While still pulling the tonsil toward the median line of the throat complete the dissection by cutting downward and medianward The tonsil is thus removed with its capsule intact The first incision separates the anterior pillar and the plica semilunaris (supratonsillar) from the anterior and superior surfaces of the tonsil The second separates the outer surface of the tonsil from the superior constrictor muscle of the pharynx The third separates the posterior pillar from the corresponding border of the tonsil The fourth incision completes the dissection by freeing the inferior attachment of the tonsil from the pharyngeal wall

#### The Removal of Fragments of the Tonsil with the Punch Forceps —

After the attempted removal of the tonsil by any method occasionally the base or a fragment of tonsil is left When this occurs it can be removed quickly and easily by means of a heavy punch forceps such as the Ruault Rhodes or Farlow The forceps should have a heavy female



FIG 192 —The Beck West method of beginning the enucleation of the tonsil is by separating the posterior pillar

blade with a wide flange to push the pillars away from the male or punch blade (Fig 193). The closed forceps should be introduced between the pillars with the cutting surfaces at right angles to the pillars as in this position they may be opened and closed without cutting the pillars. If introduced with the cutting surface of the blades parallel with the pillars the pillars may be injured or cut away. When properly placed the forceps should be pressed into the tonsillar fossa and opened and closed until the remainder of the tonsil is completely removed.



FIG 193 —The removal of a tonsil fragment with the Ruault tonsil punch forceps

When the punch forceps are removed the index finger should be introduced into the wound to search for other fragments of the tonsil. These fragments feel firm to the touch and in sharp contrast to the smooth and soft bed of the sinus. If fragments of the tonsil still remain *in situ* introduce the punch forceps and remove them or if preferred they may be dissected.

Having completed the operation mop the tonsillar fossa free of blood and search for bleeding points. If found seize them with a blunt forceps and ligate.



FIG 194 —Robertson's tonsil knife

**The Sluder Guillotine Operation**<sup>1</sup>—The *fundamental facts* underlying Sluder's technic are three in number namely (a) The guillotine will remove the tonsil with its capsule intact provided the tonsil is pushed through the fenestra of the instrument.

(b) The tonsillar fossa (bed of the tonsil) is freely movable allowing the tonsil to be dislocated forward and upward a distance of about  $1\frac{1}{2}$  inches.

(c) At a distance of  $1\frac{1}{2}$  inches anterior and superior to the tonsil is located a bony prominence on the mandible called the eminentia

<sup>1</sup> Whill's and Pylus described a somewhat similar procedure for the removal of the tonsils by the guillotine (Lancet September 17 1910)

alveolaris. This eminence corresponds to the location of the last molar tooth.

These facts are put to practical use in the Sluder operation. The tonsil is displaced forward and upward over the tubercle which in turn pushes it through the fenestra of the guillotine. The guillotine blade is then pushed home removing the tonsil with its investing capsule.

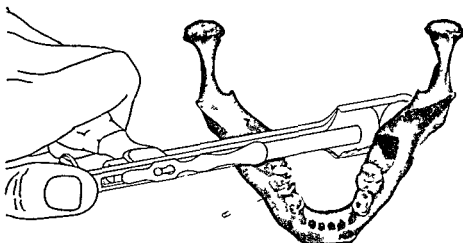


FIG. 19a. Showing the method of using the Sluder guillotine in position for the removal of the tonsil.

**Technic**—In the removal of the *right tonsil* the patient in the upright position the Sluder guillotine is grasped by its handle with the operator's *right hand* and introduced through the *left angle of the mouth* until the distal portion of the fenestral margin is in contact with the inferior and posterior portion of the tonsil.



FIG. 19c.—Tonsil removed by the guillotine (Sluder).

The instrument is then pressed firmly against the tissues which are drawn forward and upward for a distance of about 1 inch. The tonsil then rests over the eminentia alveolaris which pushes the tonsil through the fenestra of the guillotine (Fig. 199) though it may not push it all through the opening. If the blade of the instrument were forced home at this stage of the procedure the tonsil would not in all

probably be removed in its entirety, with its capsule intact. Instead only the superficial portion of the tonsil would be removed. To obviate this mishap the tonsil is drawn a little farther forward until the distal margin of the fenestra rests almost upon the apex of the eminence

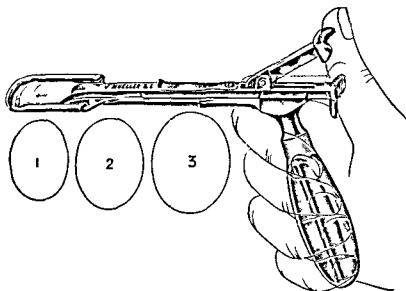


FIG. 197—Sluder's guillotine with thumb lever

The handle of the instrument is then slightly depressed to bring the inferior portion of the margin of the fenestra in firm contact with the inferior portion of the tonsil. This leaves the tonsil exposed to view (Fig. 198). The left index finger is then used to push the tonsil through

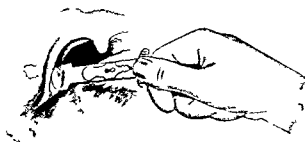


FIG. 198—Sluder's tonsil operation. First step. Placing the fenestra behind the tonsil and in front of the posterior pillar

the fenestra (Fig. 200). The blade of the guillotine should however be gently pressed against the anterior portion of the tonsil to hold it in position while the balance is being pushed through the fenestra with the tip of the left index finger.



In the third step of the operation the remainder of the tonsil is pushed through the fenestra with the tip of the left index finger. As the tonsil continues to pass through the fenestra the blade of the instrument is advanced by gentle pressure with the thumb of the right hand.



FIG. 199—Second step the tonsil is located forward over the alveolar eminence.

The tonsil tissue is readily detected by the sense of touch as it is firm and nodular whereas the mucous membrane is soft thin and smooth in texture. When the tonsil is completely through the fenestra of the instrument only the smooth thin mucous membranes of the anterior and posterior pillars lie between the tip of the finger and the

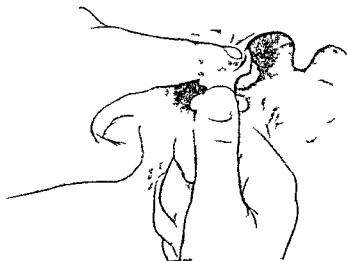


FIG. 200—Third step pushing the tonsil through the fenestra.

distal margin of the fenestra. The blade is at this time advanced until only the two mucous membranes lie between its cutting edge and the distal margin of the fenestra.

The blade is then forced home with considerable power both hands often being required for this purpose if the blade is dull.

This completes the removal of the right tonsil.

*Position of the Surgeon in Relation to the Patient*—When the patient is in the upright position, the left tonsil is removed with the guillotine grasped in the left hand, the index finger of the right hand being used to force the tonsil through the fenestra of the guillotine. In all other respects the technic is the same.

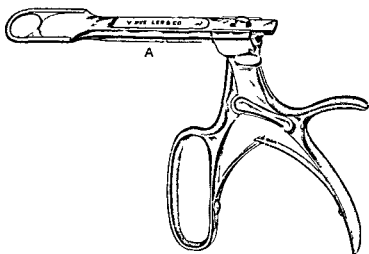


FIG. 201.—Ballenger-Sluder tonsillectome with sharp blade and scissors-handle.

**The Ballenger-Sluder Operation**—Sluder's guillotine has been modified by adding a scissors handle (Fig. 201) which greatly facilitates the work and requires very much less muscular power to cut through the tissues. The instrument is supplied in three sizes of blades.



FIG. 202.—Tonsillectomy with the Ballenger-Sluder guillotine. The tip of the instrument is placed behind the tonsil and in front of the posterior pillar. The lower pole of the tonsil is engaged first.

The tonsil is pulled against the tip of the finger instead of the alveolar eminence as suggested by Sluder. This accomplishes the same purpose and simplifies the whole procedure.

**Technic<sup>1</sup> of Ballenger Sluder Tonsillectomy**—In removing the right tonsil the guillotine is held in the right hand and inserted through the left side of the patient's mouth until the distal portion of the instrument is in front of the posterior pillar and behind and slightly below the lower pole of the tonsil (Fig 202)

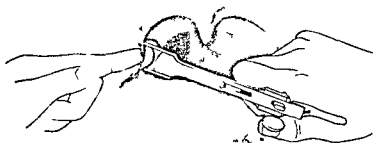


FIG 202—The guillotine is drawn forward and upward. Counter pressure is furnished by the index finger of the left hand.

The lower pole of the tonsil is now engaged in the ring of the instrument. The index finger of the left hand is placed over the anterior pillar to furnish counter pressure and by gently rocking the instrument in an up-and-down motion (upright position) the upper pole will become engaged through the ring. At the same time pressure is made by the index finger of the left hand on the tonsil (through the anterior pillar Fig 203) until the body of the tonsil is felt to slip through the ring of

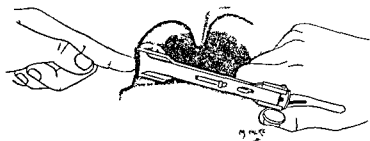


FIG 203—As the tonsil becomes engaged in the ring of the instrument the blade is gradually pushed home.

the instrument. When this occurs the entire rim of the fenestra can be felt by the finger. If a portion of the tonsil is still in the ring it can be felt as a soft mass and further manipulating is necessary.

Having pushed the tonsil through the fenestra the blade of the instrument is pushed down firmly but not severing the tonsil (Fig 204). The handle and shank of the instrument is brought at the same time over to the right side of the throat (Fig 205) and the handle of the

<sup>1</sup> The technique as given is that employed by the author (H. C. B.)

instrument is rotated upward to help lift the tonsil out of its bed and to prevent it falling into the throat as it might do if the pressure of the blade on the attachments of the severed tonsil is relaxed

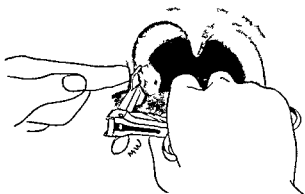


FIG. 19A. — As the blade is pushed home the handle of the instrument is brought over to the side of the mouth from which the tonsil is being removed.

Sufficient pressure should be made to sever the attachments of the tonsil. If a semi sharp blade is used the pressure from both hands is necessary. If a portion of the tonsillar attachment is still adherent after full pressure on the blade has been exerted slight traction on the instrument will finish the severing. It is never necessary to use the finger or knife for stripping the tonsil loose. If desired the blade of the instrument may be kept sharp.

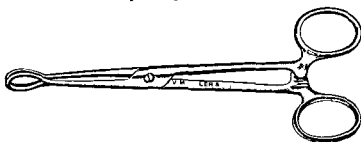


FIG. 20C. Ballenger's tonsil sponge forceps.

The left tonsil is removed as described for the right except the instrument should be held in the left hand and introduced from the right side of the patient's mouth.

In all types of tonsil operations including the guillotine it is necessary to remove the plica triangularis and the excess lymphoid tissue in the region of the plica. This may be done by grasping the plica with a forceps or hook and insinuating a snare around the mass. It may be done more quickly, easily and as effectively by means of tonsil scissors of the Metzenbaum type. The plica is grasped with a slender forceps. The convex surface of the scissors hugs the base of the tongue between

the inner surfaces of the anterior and posterior pillars completely separating all the attachments of the lymphoid mass.

The objection made to the guillotine type of instrument is that the interior pillar is sometimes cut. This objection is not based on experience, as the blade of the instrument may be made to sever the attachment of the pillar as close to the tonsil as desired indeed the tendency of the instrument is to hug the capsule of the tonsil.



1000 M. 11. 12. 13. 14. 15. 16. 17. 18. 19. 20. 21. 22. 23. 24. 25. 26. 27. 28. 29. 30. 31. 32. 33. 34. 35. 36. 37. 38. 39. 40. 41. 42. 43. 44. 45. 46. 47. 48. 49. 50. 51. 52. 53. 54. 55. 56. 57. 58. 59. 60. 61. 62. 63. 64. 65. 66. 67. 68. 69. 70. 71. 72. 73. 74. 75. 76. 77. 78. 79. 80. 81. 82. 83. 84. 85. 86. 87. 88. 89. 90. 91. 92. 93. 94. 95. 96. 97. 98. 99. 100. 101. 102. 103. 104. 105. 106. 107. 108. 109. 110. 111. 112. 113. 114. 115. 116. 117. 118. 119. 120. 121. 122. 123. 124. 125. 126. 127. 128. 129. 130. 131. 132. 133. 134. 135. 136. 137. 138. 139. 140. 141. 142. 143. 144. 145. 146. 147. 148. 149. 150. 151. 152. 153. 154. 155. 156. 157. 158. 159. 160. 161. 162. 163. 164. 165. 166. 167. 168. 169. 170. 171. 172. 173. 174. 175. 176. 177. 178. 179. 180. 181. 182. 183. 184. 185. 186. 187. 188. 189. 190. 191. 192. 193. 194. 195. 196. 197. 198. 199. 200. 201. 202. 203. 204. 205. 206. 207. 208. 209. 210. 211. 212. 213. 214. 215. 216. 217. 218. 219. 220. 221. 222. 223. 224. 225. 226. 227. 228. 229. 230. 231. 232. 233. 234. 235. 236. 237. 238. 239. 240. 241. 242. 243. 244. 245. 246. 247. 248. 249. 250. 251. 252. 253. 254. 255. 256. 257. 258. 259. 260. 261. 262. 263. 264. 265. 266. 267. 268. 269. 270. 271. 272. 273. 274. 275. 276. 277. 278. 279. 280. 281. 282. 283. 284. 285. 286. 287. 288. 289. 290. 291. 292. 293. 294. 295. 296. 297. 298. 299. 300. 301. 302. 303. 304. 305. 306. 307. 308. 309. 310. 311. 312. 313. 314. 315. 316. 317. 318. 319. 320. 321. 322. 323. 324. 325. 326. 327. 328. 329. 330. 331. 332. 333. 334. 335. 336. 337. 338. 339. 340. 341. 342. 343. 344. 345. 346. 347. 348. 349. 350. 351. 352. 353. 354. 355. 356. 357. 358. 359. 360. 361. 362. 363. 364. 365. 366. 367. 368. 369. 370. 371. 372. 373. 374. 375. 376. 377. 378. 379. 380. 381. 382. 383. 384. 385. 386. 387. 388. 389. 390. 391. 392. 393. 394. 395. 396. 397. 398. 399. 400. 401. 402. 403. 404. 405. 406. 407. 408. 409. 410. 411. 412. 413. 414. 415. 416. 417. 418. 419. 420. 421. 422. 423. 424. 425. 426. 427. 428. 429. 430. 431. 432. 433. 434. 435. 436. 437. 438. 439. 440. 441. 442. 443. 444. 445. 446. 447. 448. 449. 450. 451. 452. 453. 454. 455. 456. 457. 458. 459. 460. 461. 462. 463. 464. 465. 466. 467. 468. 469. 470. 471. 472. 473. 474. 475. 476. 477. 478. 479. 480. 481. 482. 483. 484. 485. 486. 487. 488. 489. 490. 491. 492. 493. 494. 495. 496. 497. 498. 499. 500. 501. 502. 503. 504. 505. 506. 507. 508. 509. 510. 511. 512. 513. 514. 515. 516. 517. 518. 519. 520. 521. 522. 523. 524. 525. 526. 527. 528. 529. 530. 531. 532. 533. 534. 535. 536. 537. 538. 539. 540. 541. 542. 543. 544. 545. 546. 547. 548. 549. 550. 551. 552. 553. 554. 555. 556. 557. 558. 559. 560. 561. 562. 563. 564. 565. 566. 567. 568. 569. 570. 571. 572. 573. 574. 575. 576. 577. 578. 579. 580. 581. 582. 583. 584. 585. 586. 587. 588. 589. 590. 591. 592. 593. 594. 595. 596. 597. 598. 599. 600. 601. 602. 603. 604. 605. 606. 607. 608. 609. 610. 611. 612. 613. 614. 615. 616. 617. 618. 619. 620. 621. 622. 623. 624. 625. 626. 627. 628. 629. 630. 631. 632. 633. 634. 635. 636. 637. 638. 639. 640. 641. 642. 643. 644. 645. 646. 647. 648. 649. 650. 651. 652. 653. 654. 655. 656. 657. 658. 659. 660. 661. 662. 663. 664. 665. 666. 667. 668. 669. 670. 671. 672. 673. 674. 675. 676. 677. 678. 679. 680. 681. 682. 683. 684. 685. 686. 687. 688. 689. 690. 691. 692. 693. 694. 695. 696. 697. 698. 699. 700. 701. 702. 703. 704. 705. 706. 707. 708. 709. 710. 711. 712. 713. 714. 715. 716. 717. 718. 719. 720. 721. 722. 723. 724. 725. 726. 727. 728. 729. 730. 731. 732. 733. 734. 735. 736. 737. 738. 739. 740. 741. 742. 743. 744. 745. 746. 747. 748. 749. 750. 751. 752. 753. 754. 755. 756. 757. 758. 759. 760. 761. 762. 763. 764. 765. 766. 767. 768. 769. 770. 771. 772. 773. 774. 775. 776. 777. 778. 779. 780. 781. 782. 783. 784. 785. 786. 787. 788. 789. 790. 791. 792. 793. 794. 795. 796. 797. 798. 799. 800. 801. 802. 803. 804. 805. 806. 807. 808. 809. 810. 811. 812. 813. 814. 815. 816. 817. 818. 819. 820. 821. 822. 823. 824. 825. 826. 827. 828. 829. 830. 831. 832. 833. 834. 835. 836. 837. 838. 839. 840. 841. 842. 843. 844. 845. 8

**The Ballenger-Sluder Operation Plus Dissection**—Additional dissection of the posterior pillar is advisable in some cases. Deep adhesions of the tonsil to its muscular bed are especially found in cases previously subject to quinsy or repeated severe anginas. When such adhesions are present it is difficult to drag the tonsil from the tonsillar fossa against the finger without previously dissecting the posterior pillar free from the tonsil. This may be done with scissors or knife.

Very small thin flat tonsils are not suited for removal by the Shuder method alone as there is not enough substance or bulk to the tonsils to allow the instrument to readily engage them. However if the posterior margin of the tonsil is freed sufficiently to permit the instrument to engage it the tonsil can be removed readily. It is advisable to use the smallest sized blade for the small tonsils.

### Complications and Sequelæ of Operations on the Tonsils

**Hemorrhage**—In children hemorrhage of a severe type is rare whereas in adults it is much more common on account of the larger development of the vessels and the greater abundance of fibrous connective tissue which offers more resistance to closure of the vessel.

There are five arteries supplying the tonsil all branches of the external carotid artery namely facial lingual internal maxillary ascending pharyngeal and descending palatine. The tonsillar and ascending palatine arteries are branches of the facial. The descending palatine is a branch of the internal maxillary.

Three arteries the tonsillar the ascending palatine and the ascending pharyngeal pass upward on the outside of the superior constrictor muscle which they pierce as they turn inward to ramify the tonsil and faucial pillars Just before entering the tonsil they break up into several branches (Fig 208)

The anterior and posterior pillars have arterial twigs coursing through them however the main trunks of the arterial branches are external to the palatoglossus muscle.

If bleeding points occur they should be grasped with artery forceps and ligated with a small sized plain catgut. This should be done before the patient leaves the operating room. If the patient leaves the operating room with a dry throat secondary hemorrhage very rarely occurs. If it does (and the patient has not raised his blood pressure by exertion straining etc.) a secondary infection or a preceding acute throat infection should be suspected. Many cases of persistent or recurring bleeding are due to tonsillectomies performed during the presence of or immediately after an acute nose or throat infection. Sufficient time should elapse following these infections to permit the mucosa to resume its normal color without redness or other signs of congestion.

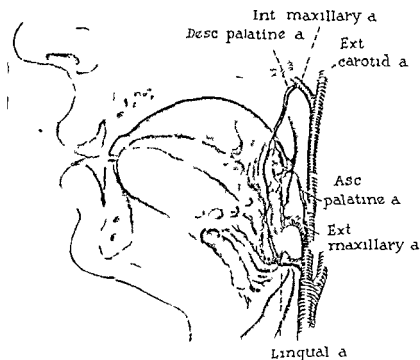


FIG 208—Blood supply of the tonsil

Late secondary bleeding usually around the fifth to seventh post operative day may be due to infection, trauma from clearing the throat coughing etc., vitamin C deficiency or some blood dyscrasia. Recent investigations have shown that an important factor in many cases is the reduction in the prothrombin of the blood induced by the administration of salicylates. The hypoprothrombinemia may be counteracted according to Nevert<sup>1</sup> by the simultaneous administration of a vitamin K like compound (synkayvite). The exact dosage of synthetic

<sup>1</sup> Arch Otolaryngol 42:14 (July) 1945

vitamin K necessary to counteract a given amount of the silicvates has not been determined as yet but according to Shapiro approximately 1 mg. of synthetic vitamin K is required to neutralize the effect of 1 gm. of acetyl silicvlic acid.

Sulfathiazole lozenges or chewing gum used three or four times daily or a 5 to 20 per cent sulfathiazole suspension sprayed into the tonsillar cavities tend to reduce the possibility of the tendency to secondary infection and inflammation of the cavities.

**Coagulation Tests** — The clinical value of blood coagulation tests prior to tonsillectomy is doubtful.

*Bleeding time* would be more valuable perhaps than coagulation time if it could be obtained with some degree of accuracy.

*Coagulation Time Technique* — A finger or the lobe of the ear is cleaned with alcohol and then coated with petrolatum. A deep puncture is made so that a drop of blood will exude without pressure onto the petrolatum covered surface. At the end of one minute and at frequent intervals thereafter a petrolatum covered needle is passed through the drop of blood until the clot can be lifted. The normal coagulation time for this method is from two to ten minutes.

Another method to determine the coagulation time is by the use of five or six capillary tubes about 10 cm. long and with a lumen diameter of from 1 to 2 mm. (about the size of the lead in a pencil). The capillary tubes are filled from the drop of blood and then one tube is filed in the center and broken into two parts every one-half to one minute until a fibrin thread appears between the fragments as they are slowly separated. The normal coagulation time by this method is from three to six minutes.

*Bleeding time* — A deep cut is made so that the blood will ooze without pressure. The drop of blood is removed every one-half minute by means of a blotter or filter paper moistened with normal salt solution. Pressure on the cut should be avoided. The time for the bleeding to stop spontaneously (normal bleeding time) is from one to three minutes. A bleeding time greater than five minutes may be considered abnormal.

**Pulmonary Abscess — Etiology** — In a study of 90 cases of pulmonary abscess made by Glowacki in the St. Louis City Hospital and other institutions he found that the incidence of pulmonary abscess was 1 in every 358 tonsillectomies.

Pilot and Davis<sup>1</sup> have shown that in lung abscess the infection seems to arise largely from the organisms usually present in the mouth and upper respiratory passages. The spirochete and the fusiform bacillus are important pathogenic agents in the production of lung abscess.

Aspiration of infected material from the tonsils is a possible and sometimes unavoidable cause of a lung abscess. The use of a suction machine during operation will do much to prevent this mishap. Many observers have shown that the majority of patients who bleed profusely under a general anesthetic for the removal of tonsils and adenoids have blood in the tracheo-bronchial tree.

Infection may also be carried to the lung by the cardio-vascular system. This is also more likely to occur during an acute upper respiratory tract infection.

It is questionable if an infection is carried from the throat to the lungs by way of the lymphatics. Most found the cervical lymphatics follow along the course of the internal jugular vein and empty into the venous system in the angle between the internal jugular and the subclavian veins.

In connection with the increased occurrence of lung abscess following tonsillectomy it should be recalled that there is an increased occurrence of lung abscess in persons who have had no operations on the tonsils.

**Symptoms**—If pulmonary abscess does follow a tonsillectomy the invasion may occur immediately or be delayed. If the invasion is delayed the patient does not do well in the interval between the operation and the first signs of invasion. The actual invasion is usually manifested by a chill or chills sensation followed by a rapid rise in the temperature which assumes a septic character. Pain in some area of the lung is usually present. Cough is a very early symptom. Odor of the breath is quite a characteristic symptom. Profuse hemipus expectoration which may become rusty as the case progresses is present as a rule. Hemorrhage may occur. Profuse sweating at night is characteristic.

The earliest physical signs are those of an infiltrated area frequently unrecognizable in the early stage becoming more clearly defined on successive days. The roentgenogram is of great help in the diagnosis.

**Differential Diagnosis**—The condition must be differentiated from tumor of the lung, bronchiectasis, chronic bronchitis with peribronchial infiltration and at times from Hodgkin's disease.

**Treatment**—In an acute or suspected case the patient should be placed on full doses of one of the sulfonamides and/or penicillin.

Postural drainage should be used to aid in the emptying of the abscess.

Bronchoscopy is useful in subacute and chronic cases if the abscess is centrally located.

In a few rare instances external drainage is necessary.

Supportive treatment should be given as indicated.

### POLIOMYELITIS

The question of performing a tonsillectomy and adenoidectomy during the period of greatest incidence of poliomyelitis in the late summer and early fall has not been decided fully at the present time. However if any question of a local epidemic is present in any community or nearby community the operation should be deferred until the epidemic has subsided or until cooler weather has arrived. Statistics show the bulbar or bulbospinal type of poliomyelitis occurs from two to three times more frequently within thirty days in tonsillectomized children than in the general population.

**Regrowth of the Tonsils**—Extratonsillar tissues often contain lymphoid tissue which following tonsillectomy in which the main tonsil mass



is completely removed are sites for future lymphoid hyperplasia in about 50 per cent of all cases. No method of tonsillectomy has been devised as yet that insures the patient against the future recurrence of lymphoid tissue in the tonsillar fossa. The cause for this postoperative hyperplasia is due to constitutional and individual factors as yet not known. This postoperative lymphoid tissue may be the site of a further focus of infection, however it frequently has no clinical significance. The regrowth occurs in children much more frequently than in adults.

### HYPERPLASIA OF THE THYMUS GLAND STATUS THYMICOLYMPHATICUS

The thymus is a ductless lymphoid gland. It is well developed in late fetal life. It reaches its highest development about the end of the second year although it occasionally remains large in the adult. After the second year it reduces in size and is for the greater part changed to adipose tissue. Roentgenograms of the thymus in young children in the tonsil and adenoid age reveal an enlargement in from 3 to 4 per cent (Schall<sup>1</sup>).

There is some doubt in regard to the causal relationship of an enlarged thymus to sudden death in infants. These cases seem to be more complex than can be explained by a purely local cause. There is usually an associated status thymicolymphaticus and also an association of other constitutional deviations and endocrine defects. Cases of suspected thymic deaths have been recorded in which the necropsy failed to reveal enlargement of the thymus. The thymus may produce signs of obstruction that may be the cause of death in some few cases. Carr<sup>2</sup> studied 520 cases of sudden death in children under ten years of age. Suffocation seemed to be the cause of death in 105 instances. In 49 of these suffocative cases pathologic changes were found in the thymus and the lymphatic system. Tracheal obstruction due to an enlarged thymus was found in 12 cases. Seven patients were classified as status thymico-lymphaticus and in 6 of these an anesthetic had been given shortly before the terminal asphyxia.

**Symptoms**—The enlarged gland may give symptoms by pressing upon the *trachea* even to the extent of reducing the anterior posterior diameter and hence would give rise to difficulty in respiration and secondarily in nursing as seen by strangling at the time of nursing. There may also be a peculiar stridor or a crowing respiration in both phases, due to the obstruction. The great vessels in the neighborhood may be pressed upon as well as some of the more important nerve trunks thus secondarily causing difficult respiration and circulation with the resultant cyanosis on crying convulsion asthma and general respiratory infections.

**Treatment**—The preoperative treatment with the roentgen ray will reduce the hazard of the subsequent operation.

<sup>1</sup> Ann. Otol. Rhinol. and Laryngol. 52: 752 (September) 1943.

<sup>2</sup> Jour. Pediat. 27: 1 (July) 1945.

## CHAPTER XXII

### DISEASES OF THE ADENOID AND LINGUAL TONSIL

#### ACUTE LACUNAR INFLAMMATION OF THE ADENOID

THE adenoid or pharyngeal tonsil consists of six fairly symmetrical folds separated by deep furrows running in a sagittal direction, which may be separated from each other like the leaves of a book. Posteriorly and sometimes anteriorly there is a curved fold connecting all of them. In the middle there is a deep fissure—the recessus medius—to which, in some instances, a blind canal leads. This fissure was formerly erroneously described as an independent structure, the bursa pharyngea, and, when infected, is known as Thornwaldt's disease.

**Etiology.**—The general etiologic factors of acute infections of the adenoid are the same as that given in "The Etiology of Acute Inflammatory Disease of the Nose, Throat and Sinuses" and will not be repeated here. It is usually associated with an acute tonsillitis with similar general and local causes.

The disease is confined largely to children and young adults due to the normal atrophy of the structure that occurs after childhood. Occasionally the adenoid persists into adult life in which event acute infections similar to acute tonsillitis may occur.

**Symptoms.**—Infection of the adenoid is rarely recognized as such on account of its hidden location back of the postnares and the soft palate. The condition may be seen, however, with a postnasal mirror. The crypts or lacunæ may be filled with a yellowish-white exudate, composed of epithelium, inflammatory exudate, and pus cocci. The secretion is often so fluid as to ooze from the crypts and coalesce with that from an adjoining crypt. At times the adenoid is only red and swollen.

The disease is usually accompanied by an initial chill, rise in temperature, swelling of the cervical lymph nodes, and a somewhat prolonged convalescence similar to that of a lacunar tonsillitis.

*Acute lacunar inflammation of the adenoid* does not occur as often as acute lacunar inflammation of the faucial tonsils. This is probably due, in part, to the absence of the deep and compound crypts that are found in the tonsil. The inflammation has a strong tendency to recur. The nose becomes obstructed and the voice quite nasal or void of resonance. There is an indefinite pain in the nasopharynx but not definitely located as when the faucial tonsils are diseased. The lymph nodes at the angle of the jaw and in the deep cervical region may be swollen and painful upon pressure.

The fever is cyclical, being less severe in the morning and greater at night. It may continue for several days and leaves the patient quite exhausted. The adenoid enlargement continues for some time, often

permanently after the fever subsides and causes more or less nasal obstruction.

To one not accustomed to examining the nasopharynx the following suggestion is of great value in making a diagnosis. If the tongue is drawn far enough forward with a tongue depressor to see behind the palatine arch the palpingo-pharyngeal fold the so-called lateral column may be found to be deeply reddened and studded with yellow follicles. This condition is characteristic of a similar condition of the adenoid.

Patients frequently complain of a feeling of fulness and pressure in the ears if the swollen adenoid presses upon the mouths of the eustachian tubes or if an associated congestion of the tubes is present.

**Treatment**—The usual local applications as given for acute tonsillitis usually irritate the nasopharynx and should not be attempted. Gargles do not reach this area. However metaplen 1 to 2000 merthiolate 1 to 1000 or a 2 per cent mercurochrome solution seem to be of value with a minimum of irritation. They should be applied directly to the infected adenoid by spraying, swabbing or dropping through the nose.

If the sulfonamides or penicillin could be given is indicated.

The patient should be kept in bed until the fever abates or a few days longer as the prostration is severe. Only a light diet should be allowed.

After complete recovery the adenoid whether large or small should be removed.

### CHRONIC HYPERPLASTIC ADENOID

**Synonyms** Adenoid vegetations pharyngeal tonsil

**Etiology**—The chief cause of enlargement of the adenoid is the irritation and inflammation which occur in the nasopharynx during attacks of acute rhinitis or one of the exanthematous fevers. The same stimulation in adults does not cause lymphoid hyperplasia to a corresponding degree.

Chronic hyperplasia of the adenoid is hereditary in many instances at least there is a *family characteristic* perhaps on account of a similar environment and similar anatomic conformations predisposing to infection of the nasopharynx.

Climate probably plays a part in the adenoid hyperplasia as a cold damp changeable climate subjects the mucosa as well as the general system to repeated shocks which lower the vitality and render the lymphoid tissue an easy prey to infection.

The condition is confined largely to children.

**Bacteriology**—The organisms in the nasopharynx that prove pathogenic for their host arranged in the order of their frequency are *Streptococcus viridans*, *Streptococcus non hemolyticus* (other than *viridans*), *Streptococcus hemolyticus* and *Staphylococcus pyogenes aureus*, *Micrococcus catarrhalis*, *Staphylococcus pyogenes albus*, *Bacillus pseudodiphtheriae*, *Pneumococcus* and *Bacillus of Friedlander*.

**Pathology**—The essential change found by microscopic examination in these enlarged adenoids is a hyperplastic one.

The normal shrinkage of the hyperplastic adenoid that usually occurs after puberty is due to a development of fibrous tissue that takes place in the substance of the adenoid hyperplasia commencing around the blood vessels invading the lymphoid tissue and replacing it. This process however may be independent of the age of the patient and is not one that necessarily commences at or after puberty but may occur at all ages.

**Symptoms**—Restlessness during the night is a prominent symptom the patient often throws the covers off during the unconscious rolling and tossing which is so characteristic of mouth breathers. Night terrors are also frequently complained of especially if the child is troubled with enuresis. They are in all probability due to reflex causes and to an excess of the half way products of metabolism.

The mental faculties are often much impaired in adenoid subjects. Approximate or difficult attention is very often present. The child is listless and has difficulty in applying himself continuously to his play studies or other tasks of which he soon tires. He has fits of abstraction. In those cases however in which there is little obstruction the mental faculties are but little affected.

The senses of taste and smell are usually impaired due to the post nasal blocking.

A chronic nasopharyngitis with frequent colds and sore throats are common.

The voice is muffled and articulation is imperfect. The resonance or timbre of the voice is greatly impaired.

Frequent ear complications are present in a majority of cases. This may take the form of a simple congestion of the eustachian tubes with slight deafness, tinnitus or vertigo or the middle ear or mastoid may become involved or a persistent purulent discharge fail to stop on account of a hyperplastic adenoid.

Examination of an advanced case reveals the open mouth, thick short upper lip and the comparatively expressionless countenance. With the laryngeal mirror or nasopharyngoscope the nasopharynx is found to be more or less filled with the adenoid mass.

Palpation reveals a gelatinous worm like mass in the nasopharynx. The finger should be anointed with vaseline before it is introduced into the nasopharynx so as to reduce its frictional qualities to the minimum. Even then great care should be exercised lest the delicate mucous membrane be injured. In spite of these precautions the finger is often streaked with blood upon its removal.

The examining surgeon should stand in front of and to the right of the patient encircling the head with his left hand and arm to steady it while the index finger of his right hand is introduced into the nasopharynx. If the thumb of the examiner is just outside the patient's right cheek he can prevent biting by pressing the thumb against the cheek wall. The soft tissues being thus forced between the patient's teeth he will not bite the examiner's finger. However if the tonsils are to be removed it is not necessary to subject the child to the digital examination as any enlargement of the adenoid can be removed at the

time of the tonsil operation. Most hyperplastic tonsils are accompanied by a hyperplastic adenoid.

The development of the face is often materially modified by the prolonged presence of an adenoid. The open mouth, the absence of the naso-labial folds, the short upper lip, the protruding and twisted central incisors of the upper jaw, the broad flat upper half of the nose and the narrow slit-like nasal openings all conspire to form the so-called adenoid face. The general expression is one of stupidity. The degree of facial disturbance varies greatly in different cases, usually in proportion to the degree of the nasal respiration rather than in the actual size of the adenoid growth.

Adenoid subjects may have a palate which is gothic or arched, especially in its anterior portion. The arch is apparently higher than normal, though the increased height is apparent rather than real. The illusion arises from the fact that the lateral diameter of the upper jaw contracts while the height of the arch remains the same, this produces a marked disproportion between its width and height.

Many individuals with a high arch are mouth breathers and have the appearance of adenoid obstruction but no adenoid is found. If a child with a high arch has its adenoid removed, the parents should be told that continued mouth breathing is probable.

The contraction of the lateral diameter of the arch sometimes causes the central incisors to protrude and to be twisted upon their axis so as to cause their posterior surfaces to face each other. The teeth are often irregular and the services of a dentist are required to regulate them.

In severe and prolonged cases the lateral walls of the chest may be contracted (Fig 209) thus throwing the ensiform cartilage into prominence. This characteristic deformity is known as pigeon chest.

**Treatment**—A hyperplastic adenoid producing symptoms should be removed. However in simple enlargement the roentgen ray or radium will reduce the size of the adenoid but will not eliminate the infection present, hence an early return of the hyperplasia is to be expected. Astringent applications are useless.

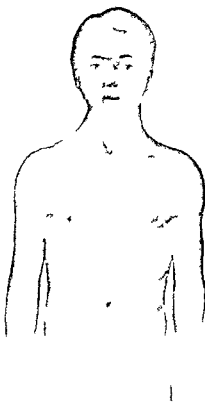


FIG 209 Deformity of the chest due to a long-continued obstructing adenoid

## ADENOIDECTOMY

An adenoid may be removed with the curette alone though this is not so thorough as other methods. A more rational and effective method is with a La Force or a Collum adenotome or some modification of them followed by a curette of the Barnhill type.

**Technic** — Nitrous oxid induction followed by oxygen ether anesthesia is preferable. If nitrous oxid is used alone sufficient time is not available to permit careful and complete technic especially proper hemostasis.

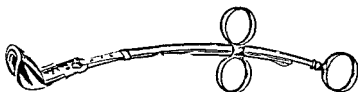


FIG. 210 — La Force adenotome

The removal of an adenoid with the La Force adenotome is performed as follows. The blade of the instrument is withdrawn leaving the fenestra open. The instrument is then introduced into the fauces the tip turned laterally engaging behind the patient's right posterior pillar. It is then turned upward into the nasopharynx. The adenoid is engaged by pushing the instrument upward and backward. The blade is then pushed home cutting the adenoid from its attachment. The instrument is then removed opened and the adenoid removed from it.

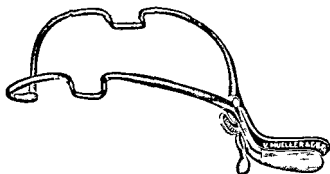


FIG. 211 — Jennings mouth gag

Introduce the curette in the same manner and engage the remaining fringe of adenoid tissue at the anterior portion of the vault just behind the posterior end of the septum as the adenotome often fails to remove the adenoid tissue in this position. The blade of the curette should be drawn forward against the septum lifted upward against the vault and then pushed directly backward until the posterior wall is reached. The blade of the curette should then be drawn downward over the posterior wall and quickly brought forward into the cavity of the mouth.

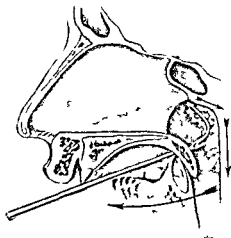


FIG. 212 — Removal of adenoid remnants with a curette. The arrows indicate the three movements necessary in a normal nasopharynx.

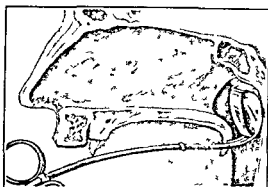


FIG. 213 — Removing an adenoid by means of an adenotome. A curette removes the remaining fringe.

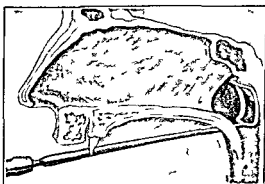


FIG. 214 — Removing the remaining fringe of adenoid tissue by means of the Barnet II curette after the mass of adenoid growth has been removed by means of an adenotome.

The tip of the suction tube should be kept in the pharynx during this procedure to prevent aspiration of the adenoid remnants

Introduce the right index finger into the nasopharynx and rub away any shreds and remnants of adenoid tissue which may remain. Also explore Rosenmüller's fossæ with the finger tip and remove the fibrous adhesive bands should any be present



FIG 215 — Barnhill's adenoid curette

### NASOPHARYNGEAL BURSA. NASOPHARYNGEAL CYST OR ABSCESS. THORNWALDT'S DISEASE

**Etiology.**—A nasopharyngeal bursa or sac seems to be a development from an embryonic pouch. The pharyngeal segment of the notochord is thought to remain united with the pharyngeal entoderm

**Pathology.**—The sac and its canal, extending to the pharyngeal mucosa, are situated beneath the adenoid or its remnants. The sac extends backward and upward to the periosteum of the occipital bone

If the canal becomes occluded a cyst forms or if infection is present an abscess may develop. An infection with an open canal would result in postnasal discharge and crusting. The crusts usually have a conical shape that fits into the conical depression of the discharging canal



FIG 216 — Pharyngeal scissors

Eagle<sup>1</sup> found the prevailing organisms to be the hemolytic staphylococcus aureus and the hemolytic streptococcus

**Symptoms.**—The symptoms usually presented are postnasal discharge, crusting, frequent colds, sneezing, hoarseness, bad taste or odor, hawking and coughing. Other symptoms may be headache or pains in various parts of the head and neck, especially in the back of the head just below the occipital bone, nasal obstruction, sore throat, nasal speech and cervical adenitis. Symptoms relating to the ear such as vertigo, tinnitus, earache and deafness may be present.

The canal may be seen by the use of a throat mirror or Yankauer's



direct speculum. If an abscess is present a small dimple may be seen on the surface of the swelling. A probe may be passed upward into it.

**Treatment**—One blade of the curved pharyngeal scissors (Fig. 217) is introduced into the canal and one half of the adenoid mass is cut. The opposite blade of the scissors is then inserted in a similar manner for the other half and that portion cut. The remaining portions of the adenoid are then removed in the usual manner. The posterior and remaining portion of the canal wall or cyst should be thoroughly curetted to remove any remaining membrane.

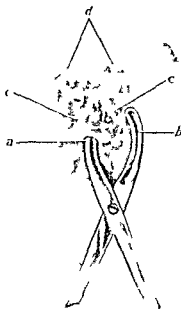


FIG. 217. Operative treatment of Thornwald's disease. *a*, the left blade of the pharyngeal scissors introduced into the suppurating sinus between the lateral halves of the adenoid; *b*, the right blade of the scissors at the border of the adenoid tissue. When the blades are closed the lateral half of the adenoid upon this side is severed. The scissors are then transferred to the other lateral half of the adenoid tissue and closed. This completely severs the lower portion of the adenoid tissue and obliterates the suppurating sinus. The remaining upper portion of the adenoid *c c d* is then removed with the adenotome and curette.

## THE LINGUAL TONSIL

The lingual tonsil may be the site of acute or chronic infections, abscesses, hypertrophy, lingual varix, hyperkeratosis, syphilis, tuberculosis, leprosy, neoplasms, benign or malignant, cysts from occlusion of the foramen cecum, accessory thyroid, and pointed foreign objects.

**Acute Congestive Lingual Tonsillitis**—Acute congestive inflammation of the lingual tonsil is characterized by no or a moderate rise of temperature, painful deglutition, and a burning, pricking sensation in the throat. There may be some tenderness on pressure in the region of the great cornu of the hyoid bone. Upon inspection the pharynx and the pillars of the fauces may be slightly reddened, while the faucial tonsils

may appear normal. The laryngeal mirror shows the masses on the lingual tonsil to be reddened and swollen.

**Treatment** — The treatment consists in brushing the inflamed masses with a 20 per cent solution of silver nitrate.

**Acute Lacunar Lingual Tonsillitis** — The symptoms of acute congestive inflammation are present and in addition the craters or crypts are lined with a whitish exudate, epithelial debris and microorganisms quite similar to the accumulations found in acute lacunar tonsillitis.

**Treatment** — The treatment consists of the local application of a 20 per cent solution of silver nitrate.

**Abscess of the Lingual Tonsil** — This process is usually characterized by a purulent accumulation beneath the lymph nodules at the base of the tongue and is usually limited to one side. The temperature is elevated and the pain upon deglutition is severe. The patient complains of soreness and great tenderness upon pressure in the region of the great cornu of the hyoid bone upon the affected side. Inspection with the throat mirror shows great swelling and redness at the base of the tongue upon the affected side. Palpation with the finger may or may not elicit fluctuation.

Phlegmonous inflammation here as in the faucial tonsil may undergo resolution without the formation of an abscess. Extension beyond the boundary of the lingual tonsil is rare. If the suppuration extends to the floor of the mouth it may be mistaken for or constitute Ludwig's angina.

**Treatment** — The sulfonamides or penicillin should be given as indicated. If fluctuation is present incision and drainage is indicated.

**Hypertrophy of the Lingual Tonsil** — Hypertrophy of the lingual tonsil is rare in children. It usually occurs between the twentieth and fortieth years of life. It is more common in females than in males. It is probably caused by repeated or continued infection of the lymph structures of the pharynx and fauces.

**Symptoms** — The symptoms are sometimes absent though the sensation of a foreign body in the throat is usually mentioned. There is a pricking sensation as though a splinter had lodged in the fauces or the patient complains of the sensation of a lump, a hair or other foreign body in the throat. Troublesome fits of coughing are often present.

During meals the symptoms frequently disappear. Pain is rarely complained of, but the disagreeable sensation already referred to is present. The use of the voice increases the symptoms and often gives rise to the pricking sensation and the cough.

Upon examination with the throat mirror a few enlarged masses are seen upon the base of the tongue. The involvement is usually on both sides but may be limited to one. The masses may be so large as to push the epiglottis backward or even to overhang it.

**Treatment** — The treatment is essentially surgical. Local applications of glycerin iodine gr xx to xxx to the ounce afford relief by reducing the swelling and sensitiveness. Linear or puncture cauterization of the masses is an effective treatment. If the lingual tonsil is greatly over-

grown it may be removed by means of Myles' lingual tonsillotome or stout curved scissors (Fig 215)

**Lingual Varix; Varicose Veins.**—**Etiology**—The exact etiology of lingual varix is not known. It occurs about equally in both sexes. Excessive and improper use of the voice may be an exciting cause. It is rare in childhood and most common between the twenty-fifth and forty-fifth years. Infectious inflammations of the pharynx and faucal tonsil and infection of the lymphoid tissue of the lingual tonsil probably are the chief etiologic factors. Some cases are reported as occurring at the period of the menopause. Constipation, high blood-pressure and an obstructed portal circulation may be etiologic factors.

**Symptoms.**—As lingual varix is usually associated with hypertrophy of the lingual tonsil, the symptoms are about the same. A sensation of scraping, burning and dryness of the pharynx may be noted.

Upon inspection, tortuous veins bluish in color, are seen at the base of the tongue partially hidden by the hypertrophied tonsil.

**Treatment**—The treatment consists in the application of the galvano-cautery to the enlarged veins and the removal of the hypertrophied lymphoid masses with the cautery point, scissors, or Myles' lingual tonsillotome. The after-treatment consists in gently massaging the wound with a cotton-wool applicator dipped in a mixture of equal parts of glycerin, tr. ferri chloridi, and tr. iodini, at intervals

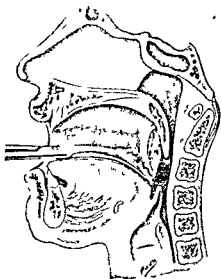


FIG 215.—Removal of the lingual tonsil with heavy scissors

of twenty-four hours. This prevents exuberant granulations, and promotes healing with a smooth wound and a minimum of cicatricial contraction.

**Leukoplakia.**—The characteristic feature of leukoplakia is the marked hyperplasia of the epithelium with some hyperkeratosis as a rule.

Excessive use of tobacco is the most common cause. It is frequently considered as a precancerous lesion.

Microscopic sections show hyperplastic epithelium with elongated papillae and an infiltrated submucosa with hyperplastic glands.

**Hyperkeratosis.**—Hyperkeratosis may occur on the lingual tonsil usually in connection with involvement of other lymphoid tissues of the pharynx. The treatment is the same as for hyperkeratosis of the tonsils.

**Tuberculosis**—Tuberculosis of the lingual tonsil is rare, but may occur as a rare complication of pulmonary tuberculosis. Small tubercles

may form which break down and coalesce forming a shallow ulcer with a dirty gray sloughing base. The congested zone found in syphilis is absent. Marked dysphagia with severe pain which radiates to the ear may be present.

**Lupus** — Lupus may be primary but usually is secondary to laryngeal or nasal involvement. Pinkish yellow nodules which tend to break down, ulcerate and then gradually heal leaving radiating scars are characteristic.

**Syphilis** — A chancre may occur on the base of the tongue, but is rare. The secondary mucous patches may appear in this area. A gumma involving the base of the tongue takes the form of a hard inflamed swelling which soon breaks down in the center leaving a punched out ulcer with red indurated edges and a dirty yellow base.

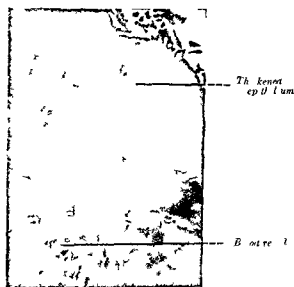


FIG. 19. Leukoplakia of the vocal cord ( $\times 30$ )

**Leprosy** — Leprosy is extremely rare and when present is secondary to involvement of other areas.

**Tumors** — Various benign tumors such as papilloma, fibroma, angioma and lipoma may originate from the lingual tonsil but are rare.

**Carcinoma and sarcoma** are less often primary in the lingual than in the faucial tonsils.

**Cysts** due to occlusion of the foramen cecum may occur in rare instances.

**Accessory Thyroid** — An accessory thyroid may occur in the mid line on the site of the foramen cecum.

Removal of the lingual thyroid may be done if the normal thyroid is present. The surgical treatment consists of the use of the actual cautery, electro-coagulation or surgical removal.

## CHAPTER XXIII

### DEEP NECK INFECTIONS

**Surgical Anatomy** The cervical fascia (fig. 220) consists of the superficial fascia and the deep cervical fascia. There are three subdivisions of the latter: (1) the enveloping layer, (2) the pretracheal layer, and (3) the prevertebral layer.

**Superficial Cervical Fascia** This fascial layer lies just below the skin of the neck. It carries the superficial vessels and nerves and is separated from the deep cervical fascia by the thin platysma muscle which extends anteriorly over the neck. This muscle arises inferiorly from the deep cervical fascia and clavicle and extends superiorly to become attached to the inferior border of the mandible. At places the platysma blends with the muscles of the face.

**Deep Cervical Fascia** The three subdivisions of the deep cervical fascia as mentioned above provide a complete envelope for all the cervical structures except the platysma muscle and the superficial blood vessels and nerves. Passing inwardly from the superficial cervical fascia the investing or enveloping layer of the deep cervical fascia is encountered and then successively the pretracheal and prevertebral layers.

The investing or enveloping layer of fascia arises inferiorly from the anterior and posterior borders of the sternum to form the suprasternal space of Burns (Gruber). These two lamellæ are then attached to the hyoid bone. Laterally the investing layer is attached to the spinous processes of the cervical vertebrae and encases the trapezius muscle. Going anteriorly from the border of the trapezius it ensheathes the sternocleidomastoid muscle and then it unites with its fellow on the opposite side above the hyoid bone as a single layer and below the hyoid with the two lamellæ that form the space of Burns. Superiorly the investing fascia is attached to the inferior margin of the mandible, the posterior angle of the jaw, the zygomatic arch (encasing the parotid gland), the mastoid process, the superior nuchal line, and the external occipital protuberance. As the single layer of fascia above the hyoid bone passes superiorly it fuses with the fascial coverings of the anterior and posterior bellies of the digastric muscles so that the submaxillary and submental areas are largely shut off from one another and from the rest of the neck.

The pretracheal layer splits off from the investing layer deep to the sternocleidomastoid muscle. It passes anteriorly to the trachea, larynx and hypopharynx. It descends into the root of the neck and mediastinum to blend with the aorta and pericardium. Laterally it ensheathes the omohyoid, thyrohyoid, sternohyoid and sternothyroid muscles and forms the carotid sheath.

The pretracheal fascia lies posterior to the esophagus, the great vessels of the neck and over the prevertebral musculature.

**Fascial Spaces in the Neck**—Three fascial spaces (Fig 221) are delineated by three layers of deep cervical fascia. The visceral space lies between the pretracheal and prevertebral fascias and contains the lower pharynx larynx trachea cervical esophagus thyroid gland and great vessels of the neck. Infections here are serious because they produced pressure on these structures as well as the ease with which extension can occur downwardly into the mediastinum laterally along the

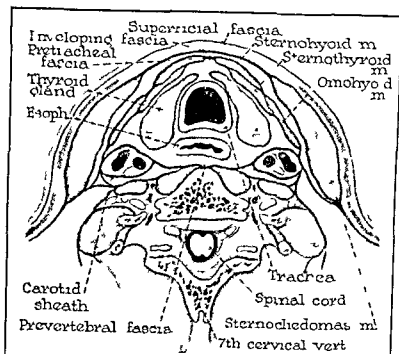


FIG 220—The cervical fascia of the neck at the seventh cervical vertebra

subclavian vessels into the axillary space or upwardly into the retro-mandibular space. The prevertebral space lies between the prevertebral musculature and the prevertebral fascia. Infections here could burrow laterally or inferiorly into the posterior mediastinum. The suprahyoid space lies above the hyoid bone between the investing fascia and the covering of the mylohyoid muscles. Infections here could break into the submental or submaxillary subdivisions of the space as described below or it could work downward into the visceral space.

The parapharyngeal space, a large, triangular compartment filled with loose, fatty tissue is lateral to the pharynx. The parapharyngeal space also known as the pharyngo-maxillary, the peripharyngeal and the lateral pharyngeal space, is composed of two compartments an anterior (prestyloid) and a posterior (retrostyloid).

The posterior compartment is formed by the carotid sheath. It

extends from the base of the skull to the visceral cervical space and contains the internal carotid artery the internal jugular vein the ascending pharyngeal artery the hypoglossal vagus cervical sympathetic glossopharyngeal and spinal accessory nerves

The anterior compartment a potential space unless infected contains connective tissue and occasionally the external maxillary arteries and usually a few lymph nodes. It extends from the base of the skull to the angle of the jaw. Anteromedially the anterior space is bound by the bucco-pharyngeal fascia covering the superior constrictor this becomes

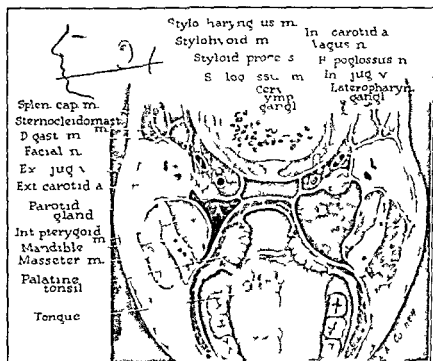


FIG. 221 The fascial planes of the neck at the level of the palatine tonsil. A the anterior (prestyloid) space. B an abscess in the anterior compartment of the parapharyngeal space pushing the superior pharyngeal constrictor muscle and tonsil to the median position.

thickened anteriorly at the pteryg mandibular ligament which is reflected onto the facial covering of the internal pterygoid muscle which forms the antero-lateral boundary. This in turn is continuous with the stylomandibular ligament (medial parotid fascia) which forms the postero-lateral wall. The posterior boundary is formed by the fascial covering of the styloid and its muscles and the anterior wall of the carotid sheath. Posteromedially the alar fascia (stylopharyngeal aponeurosis) is found. All these fascias form a complete fascia lined space except superiorly where the medial parotid layer is absent.

**Etiology**—The etiology of deep neck infections may be infections in or about the tonsils pharynx teeth sinuses thyroid gland (or its

fascia) middle ear mastoid (Bezold's abscess) petrous temporal bone base of the tongue floor of the mouth cervical vertebræ esophagus (perforation) suppurating cysts injuries of the jaw infections of the cervical lymph nodes thrombosis of the pterygoid plexus of veins and trauma. The tonsils are the most common portals of entrance usually by way of a phlebitis or a thrombophlebitis of the tonsillar veins. The cervical lymph nodes may be infected without involvement of the fascial neck spaces; however any cervical lymph node infection may result in a deep neck infection.

The principal bacteria recovered when the teeth are the source of the infection are the spirilla of Vincent. When the source is elsewhere streptococci are usually found.

**Pathology**—Once the infection has a start in the soft tissues of the neck it extends if unchecked usually by the line of least resistance into one of the fascial neck spaces. Thence it may travel upward downward or laterally following the fascial spaces as described above.

### PARAPHARYNGEAL ABSCESS

**Etiology**—The parapharyngeal space may become infected (1) by direct implantation from an operating needle (2) through the vascular channels as would occur with an endophlebitis or thrombosis and (3) through the lymphatics from suppuration of the deep cervical nodes or a lymphangitis.

Infections of this space are especially prone to follow tonsil surgery when preceded by a local anesthesia. It frequently arises as an extension from a neighboring compartment. The parapharyngeal space may become infected by way of the vascular or lymph channels from the tonsil pharynx nose sinuses adenoid pharyngeal lymph nodes cervical vertebræ mastoid or petrous bone.

**Pathology**—Deep cervical infection usually occurs as a phlegmonous type in which signs of local inflammation are predominant. A vascular form in which the local signs are subordinate but in which the symptoms are suggestive of systemic or blood stream infection is much less common.

The phlegmonous form is primarily a cellulitis within the prestyloid compartment. A later abscess formation occurs in the majority of cases. It is frequently accompanied by an unrecognized thrombosis of the internal jugular vein which may produce a fatal septicemia.

The pathogenic bacteria recovered are similar to that usually found in the tonsils and pharynx. Streptococci hemolytic and non hemolytic fusiform bacilli pneumococci and Staphylococcus aureus are the more common ones reported.

Infection may spread from the anterior compartment to the posterior compartment with extension downward along the sheath of the great vessels with a thrombosis of the jugular veins or a mediastinitis or both or the infection of the posterior compartment may extend upward along the vessel sheath resulting in an intracranial infection or erosion of the internal carotid artery infection of the anterior compart



ment may extend along the styloglossal muscle producing an abscess of the floor of the mouth (Brunner)

**Symptoms** — The symptoms of an abscess in the parapharyngeal space usually develop within four to seven days after the infection has been introduced. A high continuous temperature is frequently present especially if a lymphatic involvement occurs. Marked drops and elevations of temperature with chills are present with a blood stream complication. As a rule however the temperature is moderate not exceeding  $101^{\circ}$  or  $102^{\circ}$  F.

**Trismus** due to a splinting of the internal pterygoid muscle is a prominent symptom. It gets increasingly worse until attempts to open the mouth are very painful. Trismus may be absent if the infection is deep to the styloid process and its attached structures thereby missing the internal pterygoid muscle.

A tender swelling in the submaxillary region of the affected side especially at the angle of the mandible usually occurs before the end of the first week.

Pain in the affected area occasionally referred to the ears is a constant complaint. Dysphagia is a characteristic symptom that gets increasingly worse. Edema of the uvula, pillars and palate occurs early. Leukocytosis is usually 20,000 or more. Adenitis of the lymph nodes almost always is present but is usually overshadowed by the swelling in that region.

A displacement of the lateral pharyngeal wall usually occurs without swelling or enlargement of the tonsil. This helps to differentiate this condition from a peritonsillar abscess in which inflammatory swelling of the tonsil is present. Swelling over the region of the parotid glands is present at times. Infection lower in the neck below the level of the angle of the jaw would not give these signs.

A lateral roentgenogram of the neck shows a displacement of the trachea anteriorly especially from infections from the hypopharynx.

If the jugular vein is involved there is usually a history of a preceding sore throat with later signs of sepsis, chills and positive blood cultures. A small deep tender non fluctuating swelling may be located along its course. Emboli are not uncommon.

**Treatment** The early treatment consists of full doses of penicillin and/or one of the sulfonamides. If the infecting organism can be determined sensitivity tests can be made to penicillin, the sulfonamides and the arsenical and the best type of chemotherapy instituted.

Bed rest, hot moist dressings and large quantities of water should be prescribed.

Spontaneous evacuation of the abscess through the tonsillar fossa takes place in a number of cases usually from one to three weeks after the onset of symptoms. From an analysis of a group of 103 cases of acute suppurative conditions seen at the Mayo Clinic, Havens found an average of 22.4 days intervening between the onset and drainage of these deep neck infections. The conservative plan of treatment permitting

these abscesses to go to a stage of fluctuation before incising was most satisfactory in Havens' cases as only 1 death was reported in the 103 patients.

Surgical intervention is indicated by the signs of abscess formation and the symptoms of increasing sepsis. A reasonable length of time for the process to be walled off should be given before attempting to establish drainage.

**Intraoral Incision**—If bulging into the pharynx occurs a preliminary cocaineization is done. A curved tonsillar hemostat is inserted about one-half inch through the superior constrictor muscle into the anterior compartment and spread. The incision should be kept open until drainage stops.

**External Incision**—If the abscess points externally or shows marked swelling drainage may be established by making a small incision over the fluctuant area or over the most prominent portion of the swelling. A curved forceps is thrust into the cavity of the abscess and the opening enlarged by spreading the forceps. A counter-opening to secure dependent drainage may be advisable. A cigarette drain extending to the bottom of the abscess is sutured in place. If a large cavity is found it should be packed loosely with iodoform gauze around a cigarette drain. The gauze is removed in from one to two days, however the cigarette drain is usually left in place for about a week.

Daily irrigations of the abscess cavity with solutions of penicillin can be done through a soft rubber tube which may be used as a drain.

**Mosher's Operation**—In many instances the exact location of the pus may not be known or symptoms of sepsis may be so marked that delay in surgical intervention is not advisable or if the carotid sheath and its contents should be involved a more extensive dissection such as that proposed by Mosher should be done.

The landmarks to be kept in mind when doing the various surgical procedures on the neck are the cricoid cartilage, the tip of the great horn of the hyoid bone, the styloid process, the inner edge of the sternocleidomastoid muscle and as dissection proceeds the posterior belly of the digastric muscle.

**Technic**—Mosher<sup>1</sup> advises a T shaped incision which gives a wide exposure. The cross-bar of the T runs parallel with and close to the border of the jaw. The submaxillary salivary gland is exposed. After the facial vein is tied and cut the lower border of the gland is elevated. The finger is inserted beneath the gland and carried backward and upward until the stylomandibular ligament is felt beneath the angle of the jaw. The finger is carried upward along this ligament until the styloid process is felt. The parapharyngeal space is located by inserting the finger upward and external to the styloid process to the base of the skull. With pus in the floor of the mouth or at the base of the tongue an incision is made in the center of the floor of the submaxillary fossa and carried forward or backward according to indications.

<sup>1</sup> Trans Am Acad Oph and Otolaryngol 1929

A L. Beck uses a single incision running parallel to the border of the jaw. The deep fascia is exposed at a point behind the angle of the jaw. A closed blunt artery forceps is inserted through the deep fascia and the opening enlarged sufficiently to give room for the introduction of a finger. The finger is passed beneath the angle of the jaw to the stylo-mandibular ligament where the fascial spaces are entered as indicated.

The carotid sheath lies beneath the styloid process and may be drained at this point if necessary.

### MEDIASTINITIS

The most serious complication of deep neck infections is a mediastinitis which as a rule is manifested by visible extension of the cervical swelling.

The paths of infection to the mediastinum pass along the deep fascial planes of the neck by way of the sheath of the great vessels by extension to the retropharyngeal space (rare) and then descending by way of the prevertebral space to the posterior mediastinum along the esophagus (visceral space) to enter the mediastinum posterior to the sternum.

The signs and symptoms of mediastinitis are high fever, restlessness, rapid pulse, tenderness along the course of the great vessels and stiffness of the neck. Spasmodic alternations of the pulse and respiration are suggestive.

Jugular thrombosis has been reported in a few cases. Edema of the larynx may occur requiring a tracheotomy. Ludwig's angina, hemorrhage, osteomyelitis of the cervical vertebra and mandible, pneumonia, erysipelas, vagus involvement, meningitis, parotid abscess and septicopyemia have all been reported as complications.

### LUDWIG'S ANGINA—INFECTIONS OF THE SUBLINGUAL SPACE, SUBMAXILLARY SPACE AND SUBMENTAL SPACE

**Surgical Anatomy of the Floor of the Mouth.** In the floor of the mouth there are three important spaces (Fig. 222): the submental and submaxillary spaces below the mylohyoid muscle and the sublingual space above it. Any infection in the floor of the mouth is liable by edema to encroach seriously on the airway in the pharynx, but infections below the mylohyoid are less likely to do this because of the upward limitation to the edema; this firm, unyielding muscle exerts

**Submental Space.** The lateral borders of this space are formed by the anterior bellies of the digastric muscles; the inferior border by a line through the hyoid bone; the roof by the mylohyoid muscle and the floor by the investing fascia and skin of the chin. Infections in this space are usually confined by the firm union of the investing deep cervical fascia to the anterior digastric muscles and to the hyoid bone. A considerable edema of the chin may occur and the exploring probe may not find the pus until 2 to 3 cm. have been traversed.

**Submaxillary Space.**—The superior border of this space is demarcated by a line along the ramus of the mandible extending posteriorly until

the mastoid process is met. The posterior margin is formed by the stylohyoid and posterior belly of the digastric muscle. The anterior border is composed of the anterior belly of the digastric muscle. The roof is formed by the mylohyoid and hyoglossal muscles and the floor by the investing deep cervical fascia and skin of the neck and chin.

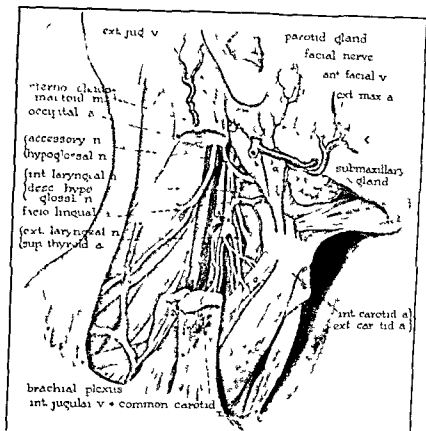


FIG. 222.—The anterior triangle of the neck. The superficial blood vessels, lymph nodes and a portion of the sternocleidomastoid muscle have been removed. The stylohyoid muscle (a) and posterior belly of the digastric muscle (b) separate the submaxillary space (digastric triangle) from the carotid triangle. The superior belly of the omohyoid muscle (c) separates the carotid triangle from the muscular triangle.

Within the space is found the submaxillary gland with its duct passing posteriorly to the posterior margin of the mylohyoid muscle to enter the sublingual space. Infection of the submaxillary space is usually confined to the space itself, but may work its way along the submaxillary duct (Wharton's) and accompanying gland structure into the sublingual space, or it may extend downward along the hyoglossal muscle to the fascial spaces of the neck.

**Sublingual Space**—This space is inclosed laterally and anteriorly by the body of the mandible. The posterior boundary of the sublingual

space is formed by the tongue the palatoglossus and styloglossus muscles and the hyoid bone. The upper boundary is the tongue itself and the floor is made up of the firm unyielding mylohyoid muscles. A potential weak spot where infections in this area may reach the submaxillary space exists at the point of entrance of the styloglossus and hyoglossus muscles near the angle filled by a portion of the submaxillary gland with its duct the glossopharyngeal and hypoglossal nerves and lingual artery and vein. With an infection in the sublingual space the edema finds the line of least resistance superiorly and posteriorly with consequent reduction of the airway.

**Ludwig's Angina.** Ludwig's angina is a rare virulent and often fatal septic inflammation of the soft tissues of primarily the sublingual space. There may be extension to the submaxillary space or to the tissues of the neck.

**Etiology.**—The etiology of Ludwig's angina has been attributed to trauma of the interior of the mouth local mouth infections dental caries especially of the molar and premolar teeth, tonsillitis and peritonsillitis trauma of dental extraction Vincent's angina facial erysipelas otitis media and externa and ulcers of the lip and nose. However infections of the tonsils and front teeth are not likely to be causative factors.

Ludwig's angina is most frequent in the young and in young adults. However no age is immune. Males are more often attacked.

Streptococci are usually found but with them are associated staphylococci bacilli coli and in some cases gas producing organisms of the anaerobic type.

**Pathology.**—The condition has been attributed to a lymphadenitis and a perilymphadenitis but it is essentially a cellulitis which spreads by continuity of tissue. The accompanying edema usually finds its easiest route of exit superiorly and posteriorly. As a result the tongue is usually edematous and displaced superiorly and posteriorly encroaching on the airway. Pus if it forms usually points within the mouth. Both the edema and pointing of the pus tend to be directed toward the mouth by the mylohyoid muscle. The infection may spread to the submaxillary space and thence to the neck by direct extension along the hyoglossus muscle.

**Symptoms.**—The duration of the infection may vary from a few days to three or four weeks. The temperature is elevated as a rule from 100° to 106° F. Leukocytosis ranges from 10 000 to 35 000 or more.

A hard board like swelling of the submaxillary and submental regions is characteristic. A swelling and induration of the floor of the mouth gums and tongue are also present. The tongue is pushed upward and backward. In severe cases the hard board like swelling may extend downward to the clavicle. Suppuration may not occur in these cases.

**Trismus** is present if the infection or cellulitis invades the parapharyngeal space causing an irritative spasm of the internal pterygoid muscle.

Abscess formation takes place in the majority of cases. Thomas reports that pus was found in 66 of his 106 collected cases.

The first complaints are usually a pain in the floor of the mouth stiffness in movements of the tongue pain in efforts to clear the throat and salivation Fever is not always present in this stage.

The displaced tongue and pharyngeal swelling may interfere with the breathing and in later stages produce asphyxia.

The constitutional symptoms are those of a severe toxemia.

There is a grave danger of loss of life from suffocation or later from exhaustion. The prognosis is grave, some writers giving the mortality as high as 43 per cent.

The infection usually extends into the submaxillary, the parotid or the pharyngo-maxillary space. Frequently all of them are involved. If the carotid sheath is invaded a jugular thrombosis may occur. Mediastinitis is common. Osteomyelitis of the mandible is a rare complication.

A type of infection resembling Ludwig's angina due to an abscess in the root of the tongue rather than under it is characterized by a tedious course with severe dysphagia and a marked systemic reaction.

**Treatment**—An attempt should be made to localize the infection while at the same time watching closely for signs of respiratory obstruction. Oxygen should be in readiness. A tracheotomy to relieve respiratory obstruction should not be delayed until the patient has exhausted himself by a struggle for air.

Penicillin and the sulfonamides should be given early and in full doses. Hot wet packs should be applied constantly to the chin and neck and hot mouth irrigations given every hour or so.

Irradiation with the roentgen ray seems to be of distinct value especially in those cases characterized by marked cellulitis.

If pus forms incision and drainage are imperative.

Incisions should be made below and parallel to the body of the mandible through the deep fascia to the depth of the submaxillary gland. The deeper exploration should be carried out with blunt forceps. An additional vertical incision should be made above the hyoid bone to the lower border of the chin. Some operators have passed the median raphe of the mylohyoids and split the geniohyoglossus muscles apart. The object of the incision is not only to evacuate pus but also to relieve tension.

Glogau advocates the procedures followed by Hajek's Clinic by making an incision along the anterior border of the sternocleidomastoid muscle and carrying the dissection by blunt or if necessary by sharp instruments well into the depth of the neck even to the mucous membrane of the pharynx.

The Mosher operation as described for parapharyngeal abscess may be done if simple incisions fail.

### CERVICAL LYMPH NODE ABSCESSSES

Suppuration of localized groups of lymph nodes are common. The superficial or deep nodes may be involved.

In inflammation of the superficial nodes with abscess formation incision should be delayed until fluctuation is present provided sepsis or evidence of deep neck involvement is absent

In inflammation of the deep nodes suppuration if present takes place late If a definite sepsis is present surgical drainage is indicated Distant metastasis and thrombosis of the internal jugular vein may occur

### THROMBOSIS OF THE JUGULAR VEIN FROM THROAT INFECTION

**Etiology**—Infection of the carotid sheath may be secondary to infection of any of the other compartments of the neck or the lymph nodes especially those located on the vessel sheath itself

The routes of infection from the tonsil to thrombosis of the jugular vein may be (1) Hematogenous A thrombophlebitis of the tonsillar veins occurs which may extend progressively until jugular phlebitis is produced (2) Lymphatic Lissenorde<sup>1</sup> believes that a lymphangitis is the primary process and that the purulent process extends secondarily to the vein causing a periphlebitis and an endophlebitis with an associated thrombosis (Stone and Berget) (3) Continuity By direct extension through continuity of tissue

The two principal hematogenous avenues are by way of the tonsillar veins into the pterygoid plexus or through the facial veins into the internal jugular vein

**Symptoms**—The signs and symptoms of sepsis are present Tenderness or swelling over the sheath may be found Torticollis to the opposite side is significant of inflammation under the sternomastoid muscle Torticollis from infection along the paravertebral and trapezius muscles is toward the same side Metastatic abscesses are common

Inflamed lymph nodes should be ruled out

**Treatment** If the carotid sheath and visceral space are involved an incision along the anterior border of the sternocleidomastoid muscle is done as for a jugular resection The sheath is opened and the jugular vein ligated and severed A gauge drum is placed in the wound

### RETROPHARYNGEAL ABSCESS—PREVERTEBRAL SPACE ABSCESS

**Clinical Anatomy**—The retropharyngeal space extends from the anterior face of the basi-occiput downward through the prevertebral space into the posterior mediastinum however the prevertebral fascia and the fascia of the superior constrictor muscle become firmly attached with the prevertebral muscles to the occiput about the level of the prominence of the second cervical vertebra This tends to confine the abscess to the upper portion

Laterally the retropharyngeal space is continuous with the parapharyngeal space

The retropharyngeal lymph nodes consist generally of two to five nodes back of the posterior pharyngeal wall near the outer edge and in

<sup>1</sup> Ztschr f Laryngol Rhinol and Otol 13 357 1925

<sup>2</sup> Arch Otolaryngol 24 141 (August) 1936

close relation externally with the great vessels of the neck. The retropharyngeal lymph nodes drain the adjacent muscles and bones, the accessory sinuses, the pharynx, the middle and internal ears and the eustachian tube.

**Etiology**—An abscess of the retropharyngeal space is not common as judged from the reports in the literature. Fuert<sup>1</sup> found a total of 157 cases of retropharyngeal abscess out of a total of 88,849 admissions to various children's hospitals as reported by various writers.

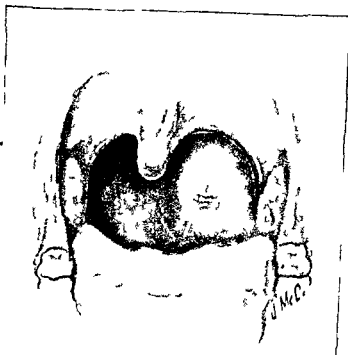


FIG. 223.—Retropharyngeal abscess on the left side.

Various factors may produce a retropharyngeal abscess.

1. Suppuration in the retropharyngeal lymph nodes occurs especially in infants and children. Babbitt reports 66 per cent occurring in the first year of infancy. Brown reports in his series 50 per cent as occurring during the second six months, and Freedman 33 per cent under one year.

An acute upper respiratory infection such as influenza, scarlet fever, measles, tonsillitis, post-tonsillectomy infection, sinusitis, especially the sphenoid or dental infections may cause a retropharyngeal abscess through infecting the retropharyngeal lymph nodes.

2. Injuries of the posterior pharyngeal wall such as may occur from foreign bodies, fishbones, etc. The injury usually causes an acute cellulitis which may spread down the loose connective tissue to the mediastinum or to the pleura.

<sup>1</sup> Ann. Otol., Rhinol. and Laryngol. 42:408 (June) 1933.



3 A retropharyngeal abscess may originate from an ear infection by direct extension of the ear suppuration from the petrous or indirectly from an extradural abscess of the middle fossa extending through the foramen lacerum or ovale. A direct extension of the infection from the ear to the pharynx by way of the suboccipital space may occur in rare instances. It may occur indirectly from an extradural abscess of the posterior cranial fossa extending to the suboccipital region. A Bezold's abscess may burrow into this space. The direct extension is more common.

4 Tuberculosis of the upper cervical vertebrae forming the so-called cold abscess.

5 Secondary to suppuration of the parotid glands.

**Symptoms**—There is usually a preceding or concurrent acute infection of the throat.

The patient if old enough complains of painful deglutition and if the swelling is marked or in the lower portion of the pharynx obstructive symptoms such as snoring, choking respiration or even dyspnea and stertorous breathing may occur. Cyanosis if observed is rarely serious.

A unilateral or predominantly unilateral cervical adenitis on the affected side is almost always observed. Cough is usually present. The voice is much the same as in quinsy. In acute cases the temperature may be elevated from  $1^{\circ}$  to  $2^{\circ}$  F. whereas in the chronic tuberculous cases little or no temperature may be present. A tuberculous retropharyngeal abscess may rupture into the pharynx producing a granuloma at the site of the rupture.

A lateral roentgenogram of the neck usually shows an anterior bulging of the posterior pharyngeal wall.

**Diagnosis**—The abscess should be differentiated from aneurysm, malformation of the vertebrae and inflammatory swelling of the mucous membrane.

Aneurysm of an artery in this region has been mistakenly diagnosed as retropharyngeal abscess, a fatal issue following the incision. The pulsation and bruit present in aneurysm should be sought for in all cases of suspected abscesses of the pharynx. The pulsation may be noted with the eye or finger while the bruit may be distinguished with the stethoscope introduced through the mouth.

Malformation of the posterior wall of the pharynx causing bulging of one side is occasionally found. The hard firm character of the mass readily distinguishes it from the soft boggy mass which is present in abscess formation.

Acute infectious inflammations of the pharyngeal mucous membrane sometimes simulates retropharyngeal abscess. The difference in the resistance upon digital examination will determine which of the processes is present.

**Prognosis**—The danger in very young subjects is chiefly due to suffocation and to strangulation upon the spontaneous rupture of the abscess. In older patients this danger is not so great as their reflexes enable them to ward it off or to anticipate it. Under treatment the

prognosis is nearly always good except when the disease is due to tuberculousaries of the vertebrae

The possible but less common results if the abscess is left to itself are: Burrowing into the parapharyngeal space with or without external pointing traveling downward behind the esophagus into the posterior mediastinum spreading by the blood and lymph streams into the meningeal area with a general septicemia hemorrhage by erosion of the great vessels in the neck pressure on the epiglottis and larynx with edema rupture and aspiration of infected contents with pneumonia lung abscess sudden asphyxia or rupture into the esophagus

**Treatment**—If seen early penicillin or one of the sulfonamides may abort an abscess formation

The surgical treatment consists of the immediate evacuation of the pus. This is sufficient in acute cases. In chronic cases secondary to

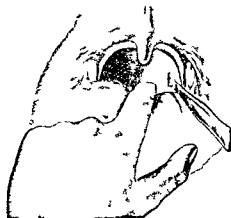


FIG. 224.—The oral operation for retropharyngeal abscess. The finger is used as a guide to the fluctuating area and as a tongue depressor while a short bladed scalpel is used to open the abscess.

tuberculosis of the cervical vertebrae this is usually done by the external operation. However the internal operation may be tried followed by the injection of iodoform glycerin emulsion (Esmarch and Kowalzig). Should simple puncture and evacuation followed by the injection of the iodoform emulsion fail the external operation may be performed.

**Technic—Internal Operation**—Place the patient upon a table with his head lowered to prevent the larynx being bathed in pus. With children this precaution is especially urgent because their reflexes are not sufficiently trained to prevent suction of the infected secretion into the trachea and lungs where it might cause aspiration pneumonia.

Introduce the left index finger into the mouth and place the tip against the soft fluctuating tumor.

Introduce a short bladed scalpel or a longer one the proximal end of which is wrapped with a strip of adhesive plaster or cotton into the mouth using the finger as a guide (Fig. 224). If the abscess is pointing a forceps may be used to puncture the wall.

Incise the abscess wall by the side of the finger. The pus then flows through the incision into the pharyngeal cavity, from which it may be removed with suction or gauze sponges or it may be expectorated by the patient. In the acute forms of the abscess recovery is rapid with little further attention necessary. In the chronic forms further attention is advisable.

An external approach may be indicated if the infection is secondary to spinal caries or if a retropharyngeal cellulitis is present. If of tuberculous origin the usual treatment of Pott's disease should be carried out.

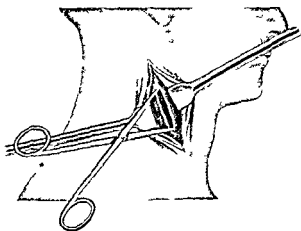


FIG. 23 — The external operation for retropharyngeal abscess. The fascia enclosing the abscess is punctured and opened with artery forceps.

*External Operation* — Generally speaking the external operation consists in making an incision either anteriorly or posteriorly to the sternomastoid muscle and extending it inwardly by blunt dissection to the anterior wall of the vertebral column where the abscess cavity is located.

If only the retropharyngeal abscess is to be included in the operation the incision should be made posterior to the sternomastoid muscle, if however, there are diseased cervical lymph nodes to be removed at the same time the incision should be made anterior to the muscle (Fig. 225).

The following steps in the operation should be observed.

The field of operation should be shaved and cleansed. General anesthesia is usually given.

An incision two or three inches long should be made through the skin over either the anterior or the posterior border of the sternomastoid muscle on a plane with the retropharyngeal abscess. The dissection should be continued until the deep cervical fascia is opened and the border of the sternomastoid muscle is brought to view.

The sternomastoid muscle is then separated by blunt dissection from the adjacent tissues and is drawn forward with a retractor to expose the operative field. Still using blunt dissection the carotid sheath with its vessels and nerves is separated from the vertebra and carefully drawn forward. The dissection is carried in front of the vertebra to the abscess wall.

The abscess wall is punctured with closed artery forceps the forceps is then introduced into the cavity the blades spread apart and with drawn from the cavity. The abscess is thus freely opened and evacuated. Digital examination of the cavity should be made for necrosed bone and to note the condition of the soft tissues and abscess contents. If the secretions are thick and caseous they may be removed by gentle curettage.

Introduce a cigarette drain into the wound. This may be withdrawn a little each day after the discharge has ceased and its use may be abandoned altogether at the end of ten days or two weeks after which the external wound closes from the bottom by granulation.

If cervical lymph nodes are to be removed or if the abscess points anteriorly to the sternomastoid muscle the incision should be made anterior to the muscle. The group of lymph nodes involved should be removed *en masse* as to leave some of them almost surely means a secondary operation.

If an anterior incision is made the thyroid gland is retracted medially. The middle thyroid vein and the inferior thyroid artery are usually ligated and severed. It may be necessary to sever the omohyoid muscle.

Blunt dissection is continued behind the thyroid gland until the esophagus is exposed. The latter is lifted away from the prevertebral fascia. A gauze drain is inserted and brought out through the lower angle of the wound.

### LIGATION OF EXTERNAL CAROTID ARTERY

Spontaneous or operative hemorrhage from malignant conditions about the head or persistent hemorrhage from other causes may demand ligation of the external carotid artery.

**Position of the Head**—The shoulders should be placed upon a block or sand cushion the chin well elevated and everted to the opposite side to expose the region of operation.

**Incision**—The incision should extend from the tip of the mastoid process close behind the angle of the jaw to the level of the middle of the larynx. At either extremity the incision is exactly over the external carotid artery. The incision should be curved medianward about 1.5 cm. as the safety of the operation lies anterior to the artery while danger lies posterior to it.

**Exposure of the Artery**—Work from below upward first exposing the superior thyroid artery which extends downward to the thyroid gland. By tracing this back to the carotid the external carotid is distinguished from the internal. Pass a chromicized catgut loosely around the external carotid. Examine the carotid and be sure that it bifurcates into the external and internal branches. If it does not it should not be ligated as the blood supply to the brain would be curtailed. In this latter event only the branches supplying the external portions of the head should be ligated the carotid being untied.

Having determined that the common carotid bifurcates as usual continue the dissection upward exposing each branch and tying it in

two places and dividing it. The dissection is thus continued upward until the level of the twelfth cranial nerve is reached and all the branches of the artery but the terminal two have been controlled. The external carotid is itself tied twice and divided between. The ligature placed loosely around the external carotid below the superior thyroid branch should not be tied until all the branches are ligated. It should not be tied sooner because the artery would collapse and render the dissection difficult. The ligature is placed in position early ready for use in case of accidental hemorrhage in the course of the dissection higher up.

The upper portion of the artery should be dissected as it passes under the transverse loop of the twelfth nerve and the co-joined stylohyoid and posterior belly of the digastric and on into the substance of the parotid gland. It should be followed to its bifurcation when possible. The dissection should be done with dissecting forceps or scissors and not with a sharp knife as it might divide some of the lower branches of the pes anserinus and cause facial paralysis or else by cutting through some of the smaller ducts of the parotid gland cause a salivary fistula (Dawbarn). Use gentle downward traction during the blunt dissection and when as high as possible seize the artery with artery forceps and tie as high above it as possible and then sever the artery below the forceps.

Close the wound by sutures leaving a cigarette drain at its lower angle or make a counter-opening  $1\frac{1}{2}$  inches below the angle and insert the drain through this entirely closing the original wound.

At the end of five or six days the drain may be discontinued and the counter-opening allowed to heal by granulation.

Structures to be avoided are the internal jugular, internal carotid, vagus nerve, the superior laryngeal nerve, the pharyngeal branch of the vagus and the glossopharyngeal nerves. They all lie behind and deeper than the external carotid artery. Careful dissection should be done.

The opposite carotid may be operated in like manner after an interval of ten days though both may be done at one time if the patient is in vigorous health. The death rate of this operation is high.

## CHAPTER XXIV

### FUNCTIONAL NEUROSIS OF THE PHARYNX

**Neuroses of Sensation**—The train of symptoms in pharyngeal neuroses of sensation is about the same as in the larynx many of them being due to reciprocal lesions (see Neuroses of the Larynx)

*Anesthesia* of the pharynx is not of any great clinical significance excepting perhaps when it accompanies progressive bulbar disease

Insane patients generally have it even though no form of paralysis is present in the pharynx or elsewhere in the body In cases of marked anesthesia involving the whole pharynx the soft palate and larynx are usually likewise anesthetic Diphtheria often causes it and sometimes it accompanies the other exanthematous fevers It may be present in local inflammations of the pharyngeal mucosa The treatment is directed to the cause

*Hyperesthesia* of the pharynx is the most frequent of the pharyngeal neuroses It often occurs in those who are otherwise healthy These cases do not tolerate the laryngoscopic mirror in throat examinations They also resist the introduction of the eustachian catheter The most sensitive areas in the pharynx are the arch of the soft palate and the vault of the nasopharynx

*Hypersensitiveness* accompanies both acute and chronic inflammation of the pharynx It is also a frequent manifestation of hysteria It is more common in men than women Habitual smokers and drinkers are subject to it It is but rarely a symptom of central brain disease The hypersensitive areas sometimes appear on the tongue

*Paresthesia* occurs about as frequently as anesthesia and less frequently than hyperesthesia and often baffles the skill of examiners and operators Tonsillar disease is often the cause of it hence these organs should be thoroughly examined for diseased conditions The passage of a bolus of food or foreign body may cause an abrasion which may be followed by the sense of a foreign body in the throat The menopause is frequently attended by perverted sensations in the pharynx Patients at this period sometimes complain of the sensation of a rope or hairs in the throat Hyperplasia of the lingual tonsil seems in some cases to cause it Granular pharyngitis especially when it involves the lateral walls (pharyngitis hypertrophica lateralis) gives rise to an irritation between the posterior pillars and the pharyngeal wall which is sometimes accompanied by paresthesia It is occasionally associated with globus hystericus

The perverted sensations complained of are cold heat a foreign body itching tickling and the dislocation of the essential parts of the fauces and pharynx The paresthesia may be so marked as to cause a distressing cough and laryngeal or esophageal spasm

*Neuralgia* of the pharynx is difficult to differentiate from muscular rheumatism. Neuralgia is not painful upon pressure while rheumatism is painful with or without pressure. Enlarged single pharyngeal follicles may become so painful as to simulate neuralgia. Localized pressure upon the follicles causes pain in rheumatic pharyngitis.

The treatment of neuralgia or muscular rheumatism should be addressed to the cause such as a focus of infection when it can be determined as well as to the relief of the pain.

**Neuroses of Motion**—Neuroses of motion of the pharyngeal muscles may like that of the larynx be divided into two general classes.

1 *Akinesis* or paralysis which may be unilateral or bilateral. The akinesis or paralysis may be still further subdivided into (a) Paralysis due to bulbar disease (central paralysis) (b) Paralysis due to diphtheria (peripheral paralysis) (c) Paralysis due to or complicating faucial paralysis (central or peripheral paralysis) (d) Paralysis of the pharyngeal constrictors.

2 *Hyperkinesis* or spasm.

**Paralysis Due to Bulbar Disease Central Paralysis**—The following central lesion may give rise to pharyngeal paralysis: acute and chronic bulbar myelitis, hemorrhage, tumors, embolism and basilar meningitis.

**Acute Bulbar Paralysis Central Paralysis**—In acute bulbar myelitis the symptoms develop rapidly, a fatal issue soon following.

The attack is sudden with severe headache, dysphagia, respiratory embarrassment, difficulty in articulation, vertigo and an unsteady gait. The prognosis is extremely grave.

The treatment is directed to the cause.

**Chronic Bulbar Paralysis Central Paralysis**—Undue exposure to cold, prolonged violent excitement, extreme fatigue and lack of nutrition are etiologic factors. Heredity seems also to largely influence its occurrence. It is more common in males than in females and is rarely observed before the age of thirty-five. In rare cases it may be due to an injury or to sunstroke. Syphilis and tuberculosis should also be included as causative agents.

**Symptoms**—Pharyngeal paralysis may be the first symptom of progressive bulbar disease. The tongue is first involved in a typical case and this is followed by paralysis of the lips and of the pharyngeal and laryngeal muscles. The paralysis at first slight gradually increases in severity.

**Diagnosis**—In the beginning the disease may be mistaken for bilateral facial paralysis though the history of a sudden onset followed by progressive chronic paralysis of the tongue, pharynx and larynx together with the lips should render the diagnosis of bulbar paralysis almost certain. In bilateral facial paralysis the tongue, pharynx and larynx are not affected. In rare cases the tongue and fauces are not involved.

**Prognosis**—The prognosis is usually grave though there may be remissions before death occurs. Patients often succumb to inanition or pneumonia.

*Treatment*—Galvanism has been used to combat the degeneration of the nerves and faradism to maintain the muscular vigor with but little success. Strychnin and arsenic are of some value. In syphilitic cases the arsenicals are indicated.

**Diphtheritic Paralysis, Peripheral Paralysis**—Paralysis of the pharyngeal muscles is often an early sequel of diphtheria and of pseudomembranous sore throat. The muscle fibers undergo more or less degeneration from the presence of the bacterial toxins and there is a mechanical hindrance from the cellular infiltration of the tissues. In addition there is a degeneration of the peripheral nerve fibers from the same causes.

*Symptoms*—The voice undergoes great changes on account of the paralysis of the pharyngeal muscles as they are utilized in articulation and voice placement. The voice has the so-called nasal quality closely resembling that present in cleavage of the hard and soft palates. The velum and uvula are relaxed and can only be raised by forced inspiration. One side or both may be affected. The paralysis occurs on or about the fifteenth day after convalescence at which time ocular symptoms may also develop.

*Treatment*—The prophylactic treatment consists in the administration of antitoxin during the diphtheria. After the paralysis has developed galvanism and faradism should be adhered to in order to maintain muscular and nervous tone while the degenerated nerve fibers are being restored.

**Paralysis of the Pharynx Complicating Facial Paralysis**—When the lesion is above the geniculate ganglion the pharyngeal is often associated with facial paralysis. The uvula does not move upon phonation and is deflected to one side. The symptoms are the same as those in diphtheritic paralysis and include such structures as are supplied by the seventh nerve.

Paralysis of the constrictor muscles of the pharynx is always accompanied by paralysis of the esophagus. The dysphagia is therefore exceedingly well marked and is often the only distinctive symptom.

**Hyperkinesis or Spasm of the Pharynx.**—*Etiology*—Spasm of the muscles of the pharynx is a rare affection. It may occur from insignificant causes as uvulitis foreign bodies globus hystericus enlarged pharyngeal follicles neuralgia and local chronic inflammations or it may be an early symptom of a serious central lesion.

The more dangerous form of spasm of the pharynx is encountered in hydrophobia edema of the glottis brain tumors paralysis agitans and other affections of the nerves.

*Symptoms*—Chronic spasm of the pharynx involving the soft palate and uvula may be the chief symptom. The levator palati is the muscle affected. The spasm of this muscle draws the soft palate upward a number of times in rapid succession after which it relaxes. During the spasm there is a clicking noise as the palate leaves the pharyngeal wall. The click is audible to those near by.



*Prognosis* —The prognosis is fair in those cases due to simple causes provided appropriate treatment is instituted. If due to a serious central lesion, hydrophobia, edema of the glottis, brain tumor, or paralysis agitans it is grave.

*Treatment* —The treatment is directed to the cause.

**Rhythmic Movements** —Continuous synchronous rhythmic movements of the palate, pharynx and larynx are rare. Leshin<sup>1</sup> found only 29 cases in the literature.

A review of these cases showed a definite relationship of these movements to organic disease of the central nervous system. No localizing neurologic significance can be ascribed to them as yet because of the varied pathology found in the few postmortem examinations made.

<sup>1</sup> Ann. Otol. Rhinol. and Laryngol. **41**: 194 (March) 1932.

# PART III

## DISEASES OF THE LARYNX

### CHAPTER XXX

#### ANATOMY—LARYNGOSCOPY

#### CLINICAL ANATOMY OF THE LARYNX

THE rigid framework of the larynx is made up of the hyoid bone, thyroid cartilage and cricoid cartilage. These rigid structures are held together by ligaments, membranes and muscles.

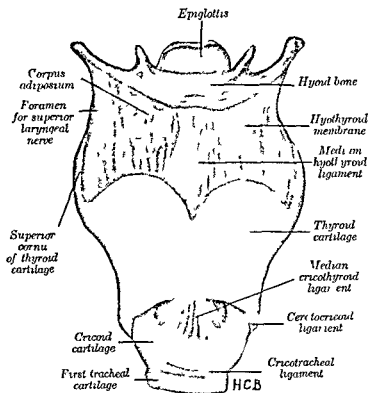


FIG. 226.—Anterior view of the larynx with its ligaments and membranes.

**Membranes and Ligaments**—The thyrohyoid membrane extends from the upper border of the thyroid cartilage to the hyoid bone. It is composed of a continuous sheet of fibrous tissue. The thickened posterior ends of this membrane reach from the superior cornua of the

thyroid cartilage to the tips of the greater cornu of the hyoid bone. This membrane consists of a central triangular portion (ligamentum hyothyroideum medium) and two lateral portions. The lateral parts are thin and lie next to the mucous membrane of the larynx. The conus elasticus extends from the superior border of the cricoid cartilage to the posterior surface of the thyroid cartilage and to the vocal process of each arytenoid. The ligamentum vocale is formed by the thickened portion of the free border.

The cricothyroid ligament forms the elastic cricothyroid membrane.

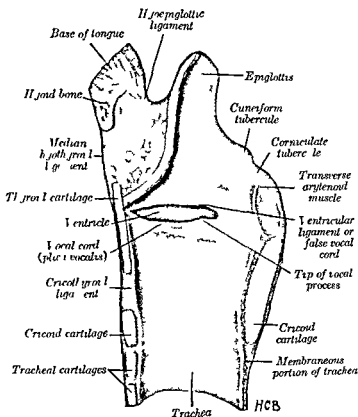


FIG. 227.—Vertical mesial section of the larynx.

**Muscles**—The muscles are divided into the extrinsic and intrinsic groups.

The extrinsic muscles responsible for the movements of the larynx may be divided into the suprahyoid and the infrahyoid groups. The suprahyoid group pull the larynx upward during the act of swallowing. These muscles are the digastric, geniohyoid, geniohyoglossus, mylohyoid, stylohyoid, and middle constrictor of the pharynx. The infrahyoid group of muscles that pull the larynx downward are the sternohyoid, sternothyroid, thyrohyoid, omohyoid, and longitudinal fibers of the esophagus.

The intrinsic muscles of the larynx may be divided into two groups, the abductors and the adductors.

Abduction is dependent on the posterior crico-arytenoid muscle which opens the glottis by rotating the arytenoid cartilages outward separating the vocal processes and the vocal cords attached to them.

Adduction is produced by the cricothyroid (tension) the crico-arytenoid lateralis and the thyro-arytenoid. The interarytenoid

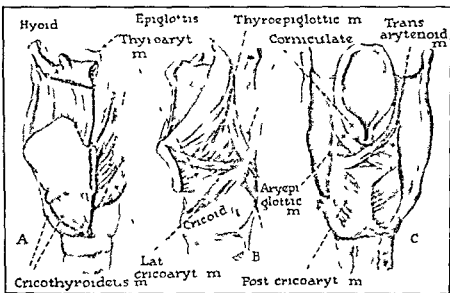


FIG. 998. Intrinsic muscles of the larynx. A, anterior view; B, lateral view; C, posterior view.

situated between the two arytenoid cartilages helps in producing adduction. The lateral crico-arytenoid muscles rotate the arytenoid cartilages inward closing the vocal cords. The interarytenoid muscle brings the arytenoid cartilages together thus closing the posterior portion of the glottis. The cricothyroid muscle puts tension on the vocal cords.

The thyro-arytenoid muscle, consisting of two parts, approximate the arytenoid and the thyroid cartilages, thereby relaxing the vocal cords.

**Vocal Cords**—The vocal cords (vocal ligaments) are triangular in shape, pearly white in color and composed of yellow elastic tissue. The upper surface of each cord is flat and forms the floor of the ventricle. Fibers of the thyro-arytenoid muscle constitute the aryvocalis muscle which is attached to the cord. The cord extends from the thyroid cartilage, near the mid-line, to the vocal process and part of the body of the arytenoid. It is continuous below with the conus elasticus. A squamous-celled epithelium covers the medial surface.

**Ventricle**—The laryngeal ventricle lies between the vocal cord and the ventricular band. The upper surface of the cord and its extension

externally forms the floor. The ventricular pouch (sacculus ventriculi laryngis) is located between the ventricular band and the thyroid cartilage.

**Mucous Membrane.**—The mucous membrane lining the interior of the larynx is a columnar ciliated epithelium except those points that come in contact which are covered with stratified squamous-cell epithelium. These portions are the vocal cords, the margins of the ventricular bands in some cases, the lingual surface of the epiglottis and the aryepiglottic folds. The laryngeal surface of the epiglottis is covered with a modified stratified columnar epithelium with a few islands of cilia.

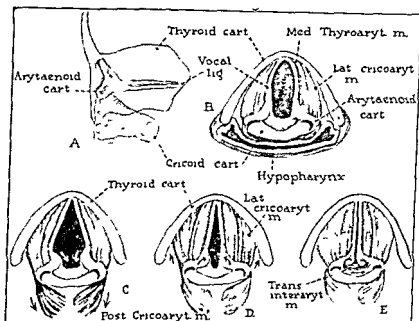


FIG. 220 — Action of the laryngeal muscles on the arytenoid cartilages and vocal cords in the acts of phonation and respiration.

**Nerve Supply.**—The motor nerve supply to the intrinsic muscles are furnished by the recurrent laryngeal nerve except the cricothyroid muscle which is supplied by the external branch of the superior laryngeal nerve and possibly the interarytenoid muscle which is thought to receive, in addition, some fibers from the internal branch of the superior laryngeal nerve. The interarytenoid muscle, being in the mid-line, receives its innervation from both recurrent nerves.

The sensory supply is furnished by the internal branch of the superior laryngeal nerve above the level of the cords. The recurrent laryngeal nerve supplies sensory fibers below this level.

**Blood Supply.**—The interior of the larynx derives its main blood supply from the superior laryngeal branch of the superior thyroid artery and the inferior laryngeal branch of the inferior thyroid artery. The

external portion is supplied by the infrahyoid and the cricothyroid branches of the superior thyroid artery.

**Lymphatics** —The interior of the larynx has two lymphatic systems an upper and a lower divided by the vocal cords. The lymphatics over the cords are very few in number. The lymphatics of the upper portion leave the larynx through the pharyngo-epiglottic folds and the thyrohyoid

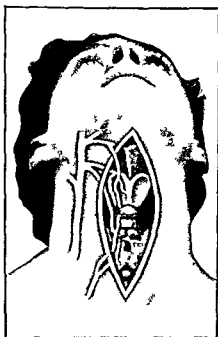


FIG. 230 —Arteries of the larynx. The superior laryngeal and the inferior laryngeal arteries, branches of the superior and inferior thyroid arteries respectively, supply the walls, lymph nodes, muscles and mucous membrane of the larynx.

membrane. The lymphatics of the lower region pass through the cricothyroid membrane. The lower lymphatics communicate with those of the trachea. The lymphatics upon the posterior wall communicate with both districts and with the lymphatics of the esophagus and the larynx.

**Piriform Sinus** —The piriform sinus or recess (Fig. 231) is part of the hypopharynx as it lies outside of the larynx proper. It is a deep depression situated on each side of the larynx external to the aryepiglottic fold and between the cricoid and arytenoid cartilages and the posterior surface of the thyroid cartilage. The sinus or recess extends superiorly to the hyoid bone and inferiorly to the lower border of the cricoid cartilage. The internal branch of the superior laryngeal nerve extends inferiorly and medially through the anterior portion of the depression. The low fold or plica produced by the nerve may be seen at times by laryngoscopic examination. A white oblique line made

by the upper border of the thyroid cartilage may be observed in some instances.

An abscess involving the thyroid cartilage frequently points in the piriform sinus (Schlugt).

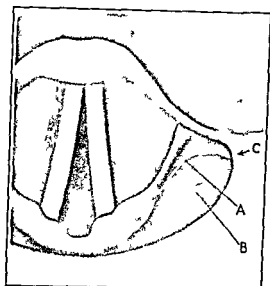


FIG. 231.—Left piriform sinus. *A* indicates the superior laryngeal nerve; *B* the white line marks the upper border of the thyroid cartilage; and *C* the pyriform gland. (Schlugt Arch. Otolaryngol.)

### INDIRECT LARYNGOSCOPY

**Technic—Preparation of the Patient**—The fauces and the larynx should be sprayed with a 2 per cent solution of cocaine to reduce the reflex irritability. The larynx is then swabbed with a 10 per cent solution of cocaine. This should be repeated at intervals of five minutes until anesthesia is induced. If this does not produce anesthesia after several applications, one or two applications of a 20 per cent solution should be made. This strength of solution should be used sparingly and with caution.

The laryngoscopic mirror is introduced into the oropharynx with its reflecting surface directed downward and forward so as to reflect the rays of light from the head mirror to the growth, the tongue being gently rolled forward on the forefinger of the left hand. The epiglottis is thereby lifted, exposing the larynx to view.

Next introduce a cup forceps or double cutting forceps (Fig. 232) into the upper space of the larynx until its cutting extremity touches the growth. It must be borne in mind that the image in the mirror is reversed; hence the movements of the instrument should be directed in an exactly opposite direction from what appears to be necessary according to the image in the mirror. For example, if the tip of the

instrument seems to need a more forward position so manipulate the handle as to move the tip backward and lower the handle. If the tip of the instrument seems to be too near the posterior portion of the image it is in reality too near the anterior portion. A little practice upon a model or upon a patient will familiarize the student with this procedure. The surgeon soon learns intuitively to move the instrument in the proper direction.

It is of great aid first to fix firmly in mind the anatomic relations of the various parts of the larynx. For example it must be remembered that the epiglottis stands at the anterior commissure of the larynx and the arytenoid prominences at the posterior commissure. These simple anatomic guides if impressed upon the memory of the operator will lead him unconsciously to guide the laryngeal instrument in the proper direction.

Having located the growth with the laryngeal forceps so manipulate the handle of the instrument as to separate the tips and then with a slight downward movement close the forceps upon the neoplasm and remove it *en masse* or in part. If the growth is large or multiple several repetitions of the foregoing procedure may be required. The growth should be removed with as little trauma to the surrounding tissues as possible.

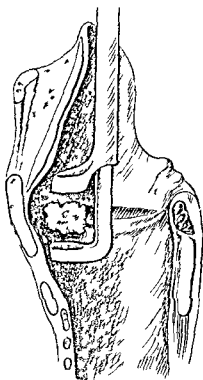


FIG. 39 Detailed drawing showing the laryngeal forceps placed to remove the neoplasm.

## DIRECT LARYNGOSCOPY

**Indications** Direct laryngoscopy is indicated in infants and young children and in adults where direct laryngoscopy does not give good visualization by the laryngeal mirror. In adults in whom the larynx is well visualized by the laryngeal mirror but some instrumental manipulation is necessary direct laryngoscopy may be advisable. Direct laryngoscopy includes the techniques necessary for the various laryngoscopes as well as for the Lynch suspension apparatus.

Direct laryngoscopy by means of the Jackson laryngoscope is described in Chapter LVIII and will not be repeated here. Direct laryngoscopy by means of the Hushinger or Atkinson laryngoscope is as follows.

**Technic**—The patient is placed in the dorsal position upon a table. The neck is inclined backward 20 degrees from the horizontal. The



head is supported by an assistant or a sand bag under the nape of the neck will act as a substitute.

Under local anesthesia in adults or general anesthesia in children the tracheo-laryngoscope (Atkinson or Haslinger's) is closed and the distal end of the spatula is inserted backward behind the dorsum of the tongue about 1 inch. The blade is slightly elevated bringing the distal end of the spatula just above the tip of the epiglottis. At this point the tip

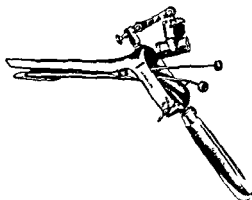


FIG. 233—Haslinger's laryngoscope.

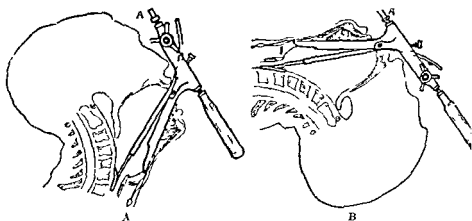


FIG. 234—Haslinger's laryngoscope illustrating instrument in use. A With patient in sitting position. B in use with patient in reclining position.

of the spatula is depressed slightly as the opposing blade is elevated by a screw. This procedure gives a view of the larynx (Figs 233 and 234).

Operation by indirect laryngoscopy may be practised when symptoms of suffocation are absent and Jackson's or Killian's tube spatula or the suspension apparatus are not at hand. The surgeon should however be prepared to perform tracheotomy if suffocation threatens during the operation.

## SUSPENSION LARYNGOSCOPY

**Anesthesia**—General anesthesia is usually employed. Induction is by nitrous oxide, oxygen and ether until deep surgical anesthesia is obtained, when a change to ether by the drop method is made. The drop method is continued while an intrapharyngeal nasal tube is inserted and connected to a gas machine for continuous delivery of nitrous oxide.

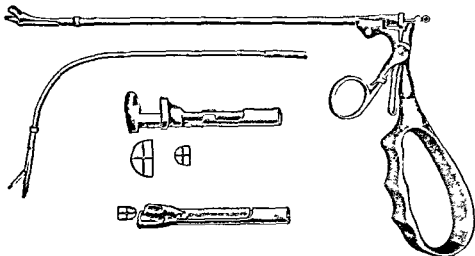


FIG. 35—Bruening's laryngeal forceps

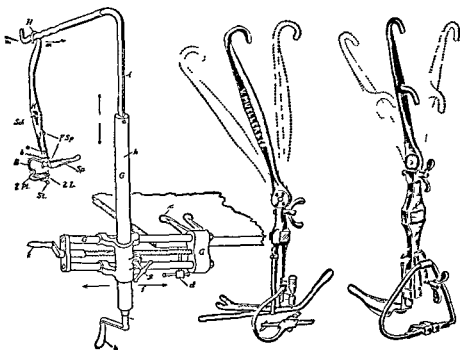


FIG. 36—A Killian's suspension gallows

FIG. 37—B Killian's suspension hooks

FIG. 38—Lynch's suspension hook

oxygen and ether through the tube. A sufficient concentration of the anesthetic mixture to abolish the laryngeal reflexes is difficult to maintain by this method over periods longer than a few minutes. Frequent interruptions to a prolonged surgical procedure may be necessary with a return to the administration of ether by the drop method. If the electrocautery is to be used the ether container should be removed from the gas machine and flushed with nitrous oxide and oxygen. To overcome these objections Adams, New, Lundy and Seldon<sup>1</sup> advocate an intravenously induced anesthesia of pentothal sodium combined with topical applications of a 10 per cent solution of cocaine to the larynx and continuous intrapharyngeal insufflation of oxygen during the intravenous anesthesia. A pre-anesthetic medication of  $\frac{1}{6}$  to  $\frac{1}{4}$  gr of morphine,  $\frac{1}{100}$  gr of atropine and  $1\frac{1}{2}$  to 3 gr of pentobarbital sodium is given.

**Technic**—The patient lies flat on the table with the head extended the crane is attached as far back on the table as is possible allowing only sufficient room to turn the handle which moves the crane horizontally the vertical position of the crane will now be on a line with the patient's shoulders. The mouth is opened wide by placing an ordinary mouth gag in the left angle of the mouth. This is steadied by the assistant who has only to keep the head extended in the middle line there being no need of support since the head is resting on the table. The gag of the hook is introduced sufficiently open to permit a view of the tip of the spatula.

Pass the spatula down along the base of the tongue until the epiglottis is seen then it is lifted with the spatula just as in bronchoscopy and by this time the short tooth plates will fall behind the teeth when the gag is opened wide to fix the tongue and epiglottis. Now the hook is placed on the crane and the worm gear joint turned to bend the hook slightly to an obtuse angle. The crane is lifted in the vertical to flatten the base of the tongue and this will raise the epiglottis so that the larynx will come into view. If the view in this position is not already sufficient then moving the horizontal crane toward the head of the table will bring the anterior commissure into view.

The growths are removed with Jackson's cup forceps.

<sup>1</sup> *Arch. Otolaryngol.* 39: 901 (March) 1914.

## CHAPTER XXVI

### MALFORMATIONS STENOSIS INJURIES DIVERTICULA CONGENITAL STRIDOR

#### MALFORMATIONS AND DEFORMITIES OF THE LARYNX

Malformations of the larynx may be either congenital or acquired. Acquired deformities are the result of postnatal disease or trauma.

*Malformations* of congenital origin are often associated with arrested development of the genitalia. The lungs, the bronchi and the trachea have the same embryologic origin (the foregut) as the larynx; hence in malformations of the larynx there may be a similar defect in these organs. In monstrosities having no larynx the lungs are frequently absent.

*Atresia* of the larynx is very rare and when present is incompatible with life.

Of the congenital deformities webs or bands across the glottis are by far the most common form.

Another form of congenital malformation consists of clefts in the interarytenoid space extending to the palate and the cricoid cartilage. The epiglottis is often deformed by arrested development, the small V-shaped epiglottis of childhood being a common variety.

*Laryngocele* (dilatation of pouches) is due to congenital malformation and failure of union in portions of the thyroid cartilage. It is rare in man though common in the lower animals.

*Esophageal pouch* or diverticulum of the mouth of the esophagus is in reality a pouch from the posterior wall of the larynx. It is seldom or never congenital in origin.

In *acquired malformations* erosions from syphilis, tuberculosis, injuries, caustics, radium, etc., may result in the partial destruction or malformation of the framework of the larynx.

#### WEBS OF THE LARYNX

Webs or bands of the vocal cords are rare. When present they usually unite the cords at their anterior portions, though the posterior portions are united at times. The bands may be at a higher level and connect the ventricular bands or they may be subglottic in position.

*Etiology*—Various theories have been advanced as to the cause of these webs or bands. It has been thought they may be due to inflammatory conditions such as congenital lues; however, there is little to support this theory. Heredity has been blamed for some cases, but this is difficult to prove. The fusion theory is the most likely cause, since in the early stages of development the primitive vocal cords are adherent to each other. A partial separation of this original fusion of the primitive vocal cords would leave webs or bands as a result.

**Symptoms** The symptoms vary with the site and extent of the obstruction. Small webs of the anterior portion of the vocal cords may cause no disturbance in phonation or respiration. Interference with the production of the voice is usually present in the larger webs. Dyspnea may be noticed on exertion. Indirect or direct laryngoscopy reveals the presence of the obstruction.

The webs are a pale color but may be differentiated from the vocal cords by their position. They may be either fragile or resilient. The perforated diaphragm variety is rare and is associated with poorly developed lungs.



Fig. 910. Web of the larynx uniting the anterior two-thirds.

**Treatment**—If causing no symptoms surgical interference is not indicated. Simple division of the web is usually followed by reformation. Bands however do not show this tendency to reform.

The web may be destroyed by diathermy followed by repeated dilations. Laryngofissure with excision of the web followed by dilations is successful in many cases.

To avoid the tendency of the webs to reform Jackson advocates incision along one cord only. The cut portion of the web will usually rest below the edge of the cord and healing will occur without adhesions. As soon as this is healed the web may be cut from the other cord and removed. Intubation tubes may be inserted after the web is excised. Repeated bouginage may be necessary. (Clerf)

## STENOSIS OF THE LARYNX

**Etiology**—Laryngeal stenosis may be due to malformations, spasmodic contractions, foreign bodies, tumors, edema, either traumatic or inflammatory, diphtheria, typhoid with perichondritis, syphilis from gumma or cicatricial changes, tuberculosis, fixation of the cords from tumors or infections, perichondritis from trauma or infections, cicatricial changes from radium, etc. hypertrophies or hyperplasias in or about the larynx, pressure from enlarged cervical lymph nodes or abscesses, bilateral laryngeal paralysis caused by bulbar lesions, tabes or thyroid operations, and from improperly performed tracheotomy or inadequate after care. Syphilis and trauma are perhaps the most common causes.

**Symptoms**—The symptoms depend upon the degree and location of the obstructing lesion. If the obstruction is extensive it may produce serious interference with respiration and phonation. A laryngeal stridor may be noticed in some cases, usually heard both on inspiration

and expiration. In congenital laryngeal stridor this is observed only on inspiration.

The diagnosis is made by inspection of the larynx with direct or indirect laryngoscopy and palpation of the cicatricial barrier with a filiform bougie. Roentgen studies should be made to rule out a foreign body.

**Treatment** — Before instituting treatment active syphilis and pulmonary infection should be eliminated as the causative factors.

If the stenosis is not too extensive some form of dilation should be tried. A preliminary low tracheotomy may be necessary in some cases. Jackson and Clerf recommend direct laryngoscopic dilation using metal bougies. Good results may be secured with progressive dilation using dilators through the tracheotomy opening. Removal of the obstructing tissue and maintenance of patency may be necessary in some cases. Clerf advocates Schmiegelow's method of treatment of cicatricial stenosis of the larynx. The stricture is removed through a laryngofissure restoring the laryngeal airway to as nearly normal condition as possible. A rubber tube is introduced through the fissure in the larynx to maintain the lumen. The tube is transfixed by means of silver wire for from three to eight weeks.

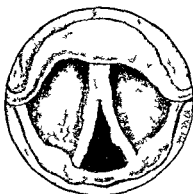


FIG. 240 Syphilitic stenosis of the larynx. The anterior third of the vocal cords is adherent.



FIG. 241 Lynah's endoscopic laryngeal bougie.

Contractions of syphilitic origin should be broken down by dilatation. The larynx should be cocaineized; the index finger of the left hand introduced through the narrowed chink of the glottis. The adhesions will thus be stretched and torn. A tube or bougie may be inserted and a larger tube or bougie introduced after leaving the first one in place a few minutes. This process should be continued three times a week until the stenosis is completely overcome. Even then the tubes or bougies should be introduced at intervals of a few weeks to prevent the reformation.

Hyperplastic or papillary growths of syphilitic origin do not always yield to the arsenicals and iodids and should therefore be removed with laryngeal forceps under general or cocaine anesthesia by either the direct or indirect method. Occasionally the papillary growths become

wedged in the chink of the glottis and cause sudden and alarming dyspnea and necessitate an emergency tracheotomy.

Tuberculous chondritis and abscess of the larynx when causing stenosis should be relieved by the removal of the diseased and dislocated cartilage with a laryngeal curette or biting forceps.

Tuberculous ankylosis of the arytenoid cartilages attended by fixation of the cords in adduction with severe dyspnea necessitates tracheotomy for the immediate relief of the symptoms or the King or Kelly operation as described for bilateral recurrent laryngeal paralysis may be necessary to create an adequate airway.

Cicatricial stenosis of lupus should be treated by dilatation with tubes or bougies as described in a preceding paragraph excepting that it may require greater persistence.

Leprous stenosis should be relieved by tracheotomy if the gravity of the suffocative attacks warrant it.

Ventricular eversion with stenosis while secondary to some diseased process of the underlying perichondrium should be overcome by removing the prolapsed sacculus membrane with a snare under cocaine anesthesia. Failing in this tracheotomy may be performed and the everted mass removed subsequently by laryngofissure.

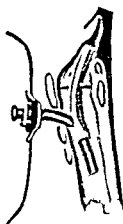


FIG 241 --Tracheotomy tube with rubber tube extension for stenosis of the larynx

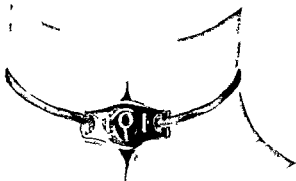


FIG 243 --Tracheotomy tube with rubber tube extension for stenosis of the larynx

Traumatic stenosis whether of chemical or mechanical origin may often be treated successfully by first performing laryngofissure and then introducing a tracheotomy tube with a rubber tube extending up and from it through the chink of the glottis (Figs 242 and 243). The

rubber tube exerts constant pressure and gradually removes the hyperplastic tissue causing the stenosis by pressure atrophy. The tube should be worn for from four to sixteen weeks and should be removed every two or three days.

### FRACTURE OF THE LARYNX

Fractures of the larynx are rare. Stimson found 67 cases reported up to 1900. It is most common in male adults probably due to the fact that the cartilages undergo calcification around the twenty-fifth year and men are more often exposed to trauma.

It is usually caused by direct violence especially from compression backward against the vertebral column. The thyroid cartilage is most frequently fractured either of the simple or compound type. The arytenoids may be loosened and the soft parts extensively involved. If the mucosa is torn emphysema may result and extend to distant parts of the body.

**Symptoms**—The symptoms and signs depend on the cartilage involved and the extent of the injury. Pain usually is present in every case and is accentuated by phonation or deglutition. Tenderness and swelling may be quite marked. Loss of consciousness is not uncommon. Dyspnea is often present in varying degrees due to edema of the soft tissues or to blood in the trachea. Emphysema with asphyxiation has been reported. A fragment of cartilage in the air passages has been reported in some cases. Hemoptysis may be present but is usually not severe. Crepitation may be elicited. A change in the outline of the neck may be noted. Roentgenograms may reveal the fracture.

**Prognosis**—The prognosis should be guarded depending on the extent and the cartilage involved. A fracture of the cricoid cartilage is more serious than of the thyroid cartilage. The mortality has been estimated around 30 per cent (Gardner<sup>1</sup>). Death usually results from suffocation due to edema of the glottis or of the vocal cords or from bronchopneumonia.

**Treatment**—In the less severe cases the treatment is palliative and symptomatic. The patient should be kept in bed with absolute rest of the voice. The room should be kept moist. An ice collar for the neck may give relief. A low tracheotomy is necessary if dyspnea appears.

Opiates may be given to relieve pain and apprehension. Fluids should be given subcutaneously or by rectum. Immobilization of the larynx is sometimes necessary to produce union.



FIG. 244. Fracture of the thyroid and cricoid cartilages and the hyoid bone from manual strangulation.



### PROLAPSE OF THE VENTRICLE OF MORGAGNI

An actual prolapse of the ventricle does not occur but that which appears to be a prolapse is in fact an infiltration of the tissues. The pink fleshy tumor like mass is quite soft to probe pressure.



FIG. 215.—Prolapse or erosion of the left ventricle. The tumor formation extends above the vocal cord and below the ventricular band.

The etiology is unknown. Many of the reported cases have been attributed to syphilis or tuberculosis.

The treatment by local applications is useless. A number of observers have reported successful though fugitive replacement of the pouching membrane. Replacement followed by cauterizations to excite inflammatory reaction offers some hope of permanent cure. Extirpation of the mass with cutting forceps or by thyrotomy may be resorted to if simpler measures fail. Antisyphilitic remedies should first be tried however in all cases in which syphilis is thought to be an etiological factor before surgical interference

is attempted. In some instances it becomes necessary to perform a tracheotomy to relieve suffocative symptoms.

### DIVERTICULUM OF THE LARYNX LARYNGOCELE

Diverticula or laryngocle of the larynx (air tumors) are rare. Videbeck found reports of only 60 to 79 cases of laryngocle. They may be extra or intralaryngeal or both. The true form is probably congenital and may be analogous to the lateral air sacs found in howling monkeys. The diverticula may start from the interior of the upper segment of the larynx and extend above the ventricular fold or they may be appendices of the laryngeal ventricle placed between the ventricular fold and the inner surface of the thyroid cartilage. The sac may pierce the hyothyroid membrane and pass into the neck. Chouke<sup>2</sup> reported 2 cases which terminated near the anterior tips of the great cornu of the hyoid bones.

The acquired or symptomatic type usually arises from a chronic granuloma such as that associated with syphilis or tuberculosis. Partial closure of the ventricle of Morgagni occurs with dilatation of the ventricle upward or downward following excessive blowing phonation coughing etc.

### DIVERTICULUM OF THE ESOPHAGUS

Diverticula of the esophagus may occur in two different ways. They may be the result of pulsion or pressure within the esophagus or pharynx

<sup>1</sup> Acta oto-laryngol. 29 123 1941

<sup>2</sup> Arch. Otolaryngol. 16 538 (October) 1932

or they may follow traction from without. Both types would seem to require a weak muscular wall.

**Pulsion Diverticulum** The pulsion diverticulum or as it is more correctly called the pharyngo-esophageal diverticulum comprises over 90 per cent of all esophageal diverticula. It usually occurs in males past middle life. The occurrence of congenital diverticula is doubtful.

The pouch is formed by a protrusion of the mucosa between the transverse and oblique fibers of the inferior constrictor muscle. This always occurs in the posterior mid line. As the pouch increases in size it descends and tends to be deflected to one side (usually the left) of the mid line. It is located as a rule between the prevertebral and pretracheal layers of the cervical fascia. The wall of the sac varies from extreme thinness to great thickness. Retained liquids and foods are always present.

**Symptoms** — The majority of the pulsion diverticula present a history of difficulty in swallowing and regurgitation of foods. Cough and huskiness of the voice may result from pressure on the recurrent laryngeal nerve. Horner's syndrome consisting of a unilateral sweating, flushing or pallor of the face and a unilateral ptosis, myosis or mydriasis may be present.

A gurgling sound may be produced by pressure of the hand on the side of the neck if tried immediately after eating (Boyce's sign).

**Diagnosis** — The diagnosis is made from a roentgenologic study using bismuth or a suspension of barium. The diagnosis is confirmed by esophagoscopy. Prior to esophagoscopy the patient should swallow a string with a shot to act as a guide in passing the esophagoscope.

**Surgical Treatment** — The surgical treatment of pulsion diverticula may be a two-stage procedure or a one stage operation.

**Technic** Combined vertin and local anesthesia is used.

The incision is made along the anterior border of the sterno-cleido-mastoid muscle from the level of the hyoid bone to one inch above the sternum exposing the anterior belly of the omohyoid muscle.

The common carotid and internal jugular vein are exposed and retracted outward. The sheath covering the trachea and the esophagus is incised. The trachea is retracted to the mid line.

The esophagoscope is introduced and the sac emptied of its contents. The sac is grasped and drawn upward and outward. When the dissection is complete the esophagoscope is reintroduced after threading the swallowed string through the esophagoscope.

The sac is then transfixed and severed close to the esophagus. The stump is inverted by reef sutures.

The musculature over the sac is sutured to give additional protection against leakage and the esophagoscope is removed after passing a duodenal tube through the esophagoscope into the stomach and up through one nostril.

The incision in the neck is closed and soft drains are placed below the deep fascia.

Feeding through the tube should continue for about two weeks when soft foods may be given by mouth for another three or four weeks.

**Traction Diverticulum** — Traction diverticula are usually found in the middle third near the bifurcation where the bronchial lymph nodes may cause adhesions. It is usually located in the anterior or lateral wall of the esophagus in an oblique and upward position. They are usually found at autopsy.

As a rule they are without clinical significance as they seldom give rise to symptoms. Perforation may occur in the pleura, lungs, mediastinum or pericardium. Double diverticula have been reported.

The diagnosis can be made from a roentgenologic study using bismuth or a suspension of barium followed by esophagoscopy.

### DIVERTICULUM OF THE TRACHEA

A diverticulum of the trachea may occur but is very rare. The tracheal diverticulum may be congenital or acquired. The acquired tracheal diverticulum has been attributed to an infection in the mucous glands of the posterior tracheal wall with subsequent development of a *diverticulum* (Zeigler<sup>1</sup> quoting Chiari). The immediate exciting cause would seem to be increased intratracheal air pressure from exertion, horn blowing, etc.

The differential diagnosis must be made from an esophageal diverticulum and a tracheal retention cyst. The instillation into the trachea of opaque oil with the patient in the modified Trendelenburg position might assist in the diagnosis.

The symptoms would be those from a tumor formation with disturbance of the voice and cough.

Excision of the diverticulum by methods similar to esophageal diverticulum would seem to be the treatment of choice. Injury to the recurrent laryngeal nerve should be avoided.

### CONGENITAL LARYNGEAL STRIDOR

Congenital laryngeal stridor should be limited to those cases in which symptoms occur from an exaggeration of the infantile type of larynx.

An exaggerated form of the infantile larynx is found in which the epiglottis is excessively curved or folded. The epiglottis has been described as beak shaped. The condition has been attributed to various congenital malformations exaggerated by a laxity of the tissues. A narrowing of the laryngeal orifice is usually present.

Some authors believe the stridor may be of central origin due to cranial injuries at birth without laryngeal deformity.

The stridor occurs on inspiration at or shortly after birth. It is increased when the child is active and may disappear when quiet. Other voice sounds are normal. Cyanosis is rare. As the larynx develops the stridor tends to disappear as a rule during the second year. Death from suffocation has been reported usually associated with an intercurrent respiratory infection.

## CHAPTER XXVII

### ACUTE INFLAMMATORY DISEASES OF THE LARYNX

#### ACUTE CONGESTIVE LARYNGITIS

**Synonyms** — Acute catarrhal laryngitis simple laryngitis angina larynx

Acute congestive laryngitis is an acute inflammation of the laryngeal mucosa and of the vocal cords. It is characterized by hoarseness or aphonia and occasionally pain upon phonation.

**Etiology** — The etiology of acute congestive laryngitis is the same as has been considered in discussing The Etiology of Acute Inflammatory Diseases of the Nose Throat and Sinuses. The process is usually an extension to the larynx from a similar acute infection of the nose sinuses or throat.

It is more common in the winter months than in the summer. Men are more frequently affected than women. It presents special features in the young which will be considered separately.

The acute infectious fevers such as influenza measles scarlet fever whooping cough typhoid and smallpox may result in an acute laryngitis. It may be part of a syphilitic or tuberculous infection.

Excessive use of the voice is a primary factor in the etiology in many instances especially if an acute throat infection is present.

Irritating fumes acids and chemicals as well as certain powders dusts etc. may be the etiologic factor.

**Pathology** The histologic changes in acute congestive laryngitis are the same as in inflammations of the mucosa of other portions of the upper respiratory tract. The peripheral vessels are congested and the tissues are infiltrated with round cells and leukocytes. If the inflammation runs a short course the infiltration disappears leaving little or no trace of its occurrence. The secretions at first thin and scanty later become heavier and more profuse. In severe cases they may become purulent and streaked with blood from the superficial follicular ulcers.

**Symptoms** The outstanding symptom is hoarseness with occasional pain and cough. The voice may be hoarse in any degree or aphonia may be present. The hoarseness is due to the swelling and infiltration of the cords and adjacent mucous membrane.

The character of the cough depends largely upon the individual though it bears some relationship to the stage and intensity of the disease. In the early stage it is usually soft and husky whereas later it is more heavy and harsh. In those cases in which there is extensive infiltration and edema it is spasmodic hoarse and wheezy with but little tonal quality. If the inflammation is limited to the interarytenoid space hoarseness may be absent.

In cases complicated by excessive edema the respiration may be labored because of the edematous swelling.

The temperature varies from normal or a slight elevation to one of several degrees according to the severity of the inflammation and the virulence of the microorganisms contributing to the phenomena.

Examination with the laryngeal mirror and reflected light shows the mucosa to be red and more or less swollen from hyperemia and infiltration or edema according to the virulence of the inflammatory process. The cords are pinkish red or even as red as the mucosa. Sometimes ecchymotic spots of extravasated blood may be seen on their upper surfaces or free borders. The secretions at first thin and scanty later become thick, semitranslucent or opaque according to the amount of lymphocytes thrown out. They have a tendency to accumulate at the anterior commissure and to some extent along the cords. They appear as opaque plugs rather than as thin diffused glairy masses.

When follicular ulcers are present the denuded areas appear as slightly roughened red spots or if covered with secretions as whitish opaque ones. In some cases there is a cloudy swelling of the epithelium in isolated areas. These areas are the beginnings of ulcerations (rare). They appear as slightly elevated patches with a grayish semitranslucent covering. Hemorrhages may occur at the commissure of the cords or on the ventricular bands. At first the site of the hemorrhage is red later almost black. When the inflammation is severe the venous flow may be blocked so that the parts are edematous.

**Treatment** — The treatment consists largely in giving the voice complete rest. The patient should be confined to his room the temperature of which should be maintained at from 67° to 70° F. The atmosphere may be surcharged with steam from boiling water to which the tincture of benzoin compound has been added. 1 tablespoonful to the pint of boiling water. Instead of steaming the room the patient may inhale the benzoin steam from a croup kettle several times a day.

A compress of cold water applied over the larynx beneath a flannel bandage gives relief in most instances.

For the relief of the cough codeine sulfate gr  $\frac{1}{12}$  to  $\frac{1}{2}$  may be administered every two or three hours until relief is afforded.

### ACUTE LARYNGITIS IN CHILDREN

**Synonyms** — Pseudocroup, false croup, Miller's asthma, laryngitis stridulosa.

In children acute laryngitis is often characterized by a spasmodic croupy or barking cough and suffocative attacks.

**Etiology** — The etiology of acute laryngitis in children is in general like that of acute laryngitis in adults. In children the bulk of the glottis is both relatively and absolutely smaller, the lymphatic and vascular structures are more abundant and the mucosa is more loosely attached to the underlying tissues.

**Symptoms** — The symptoms of acute laryngitis in children resemble those of the adult type though in many cases the spasmodic suffocative attacks are present on account of the extreme swelling and edema of the subglottic tissue

Examination reveals the swollen mucosa beneath the true cords seen through the chink of the glottis as beefy red bands

**Diagnosis** — Acute laryngitis in children should be differentiated from diphtheria and foreign bodies in the larynx

*Diphtheria* is characterized objectively by a membranous deposit which may be seen upon laryngoscopic examination. It may be either on the laryngeal mucosa or in the trachea or both. Cultures show the diphtheria bacilli. In acute laryngitis there is an absence of the false membrane and the bacilli while the mucosa is greatly swollen and red dened. If it is of the subglottic variety the swollen red mucous membrane may appear as round reddened cords parallel with and below the true cords. The temperature is usually higher in acute laryngitis in children than in true diphtheria while the prostration is not so great.

*Foreign bodies* in the larynx are differentiated by the history of the accident the sudden onset of the suffocative symptoms with no prodromal history and the roentgenologic and other evidence of a foreign body in the larynx

**Treatment** During the acute stage the child should be confined in a room kept at a temperature of about 70° F and the atmosphere surcharged with steam. If there is much mucus in the throat and trachea suction should be used. If the secretions are scanty or tenacious the inhalation of steam with one tablespoonful of tincture of benzoin compound to the pint of steaming water will stimulate the secretions and give marked relief.

The external application of an ice-bag or a cold compress to the neck often affords relief.

In the *later stage* paregoric codeine etc may be administered in small doses to relieve the cough.

### ACUTE LARYNGO-TRACHEO-BRONCHITIS

**Etiology** — Acute laryngo-tracheo-bronchitis with dyspnea occurs in infants or young children following an acute infection of the upper respiratory tract. It may be due in some cases to a foreign body in the trachea or bronchi.

In the severe form the predominating age is from twelve to twenty four months. It is seldom seen after the age of seven years. In young children the excess of loose areolar tissue in the subglottic area permits an edematous inflammation to form and mechanically shuts off the trachea. It is more commonly found in boys than in girls. It has been reported in epidemic forms especially following influenza.

*Streptococcus hemolyticus* and *viridans*, *Staphylococcus aureus* and *albus*, *pneumococcus* and *Micrococcus catarrhalis* are the organisms

usually responsible. The streptococcus is recovered in about 90 per cent of the cases. Mixed infection is present frequently.

**Pathology** — The pathology consists of inflammation, swelling and edema of the subglottic area with redness and crusting of the laryngeal tracheal and bronchial mucosa.

The subglottic tissues show semielliptic folds, one below each cord due to the inflammation and edema of the loose connective tissue in the conus elasticus. A thin exudate may be seen which can be wiped away without leaving an eroded or bleeding surface. Ulcerations or membrane as occur in diphtheria are absent.

The vocal cords may be slightly swollen and reddened. At times a gray pseudomembrane may be present.

There is usually a thick tenacious discharge in the trachea and bronchi made up of fibrin, leukocytes, epithelial cells and organisms. Some tracheal orifices are filled with pus and other orifices are obstructed with straw-colored or brownish crusts. A dry or glazed appearance of the tracheobronchial mucosa may be seen at times. The bronchi are obstructed by inspissated secretion which the cough reflex is unable to expel.

**Symptoms** — Hoarseness is the outstanding symptom but may not always be present. Dysphonia often progresses until a stage of complete aphonia is reached. A dry croupy cough, non-productive in character is present as a rule. The temperature as a rule is from 103° to 106° F. More or less dehydration occurs due to the unwillingness or inability of the child to take sufficient fluids. The red blood cells and hemoglobin are not far from normal. The white cell count is usually within the normal range but in some instances it may reach 10,000 to 15,000.

Dyspnea is a late symptom usually due to swelling or crusts in the subglottic region. This mechanical obstruction is manifested by an indrawing on inspiration of the suprasternal notch, the epigastrium and the intercostal and supraclavicular spaces. Restlessness accompanied by an anxious or frightened look comes with the effort to obtain air. The child is constantly changing its position in this attempt.

The differential diagnosis should be made from laryngeal diphtheria, foreign body, the various forms of laryngitis, obstructing tumor, abscess or laryngeal stenosis from any cause.

**Prognosis** — The prognosis depends to a large extent upon the dyspnea that may be present, especially in the very young patient. If the dyspnea is long continued, cardiac exhaustion may ensue. The advent of bronchopneumonia is a factor of importance in the prognosis. A mortality of 70 per cent has been reported in children under three years of age before the advent of the antibiotics and the sulfonamides. Since then the incidence and the mortality have dropped. The prognosis is better in older children.

**Treatment** — The patient should receive full doses of penicillin or one of the sulfonamides. A steam tent or preferably an entire room should be kept at a temperature of 70° F. with the humidity near 70 to 80 per

cent saturation. This may require the use of ice cakes and mechanical vaporizers rather than hot steam. A high fluid intake is desirable. Oxygen should always be available if any evidence of cyanosis appears.

The peroral or tracheotomy aspiration of secretions or forceps removal of crusts is necessary in many of the severe cases. Intubation is seldom indicated as it does not relieve the laryngeal obstruction.

If dyspnea is present with sternal and intercostal retraction with or without cyanosis tracheotomy should be done. A local anesthesia is employed. The tracheotomy may be preceded by bronchoscopy leaving the bronchoscope in place as it is often difficult to locate the trachea in an infant. The inner tracheotomy tube should be removed and cleansed frequently. It is necessary to use suction through the tracheal opening in many cases to remove the thick heavy tenacious mucus. The tracheal tube may have to be left in place for as long as three weeks or more in a few cases. If plugs of mucus form bronchoscopic drainage is indicated.

### MEMBRANOUS LARYNGITIS

**Synonyms** —Croup pseudomembranous croup streptococcic or pneumococcic membranous croup.

**Definition** —Membranous non-diphtheritic laryngitis is rare. It is characterized by an inflammation of the larynx attended with the formation of a false membrane of non-diphtheric origin. Opinions differ as to the unity or duality of this disease and true diphtheria. The evidence however seems to show that they are two diseases the latter being due to an infection from the Klebs-Loeffler bacillus while the former is due to an infection from other microorganisms usually the cocci or to a caustic irritant. When due to the latter the membrane is not of microbic origin though it may become infected secondarily. Under the microscope it presents the same appearance as that due to cocci.

**Etiology** —The causes of membranous laryngitis are microbic chemical and mechanical irritants. Exposure to damp and cold are predisposing causes in young children. The cases of microbic origin usually follow or attend scarlet fever measles smallpox etc. In rare instances the pseudomembrane of Vincent's angina extends into the larynx producing a membranous laryngitis. It is essentially a disease of young childhood occurring chiefly between the ages of two and eight years. It is most prevalent in the winter season.

**Pathology** —The membrane is in two layers a superficial or epithelial and a deeper or fibrous layer. It is comparatively loosely attached to the mucous membrane whereas in diphtheria it is firmly attached. The membrane is not grayish white as it usually is in diphtheria but is yellowish and of a soft friable consistency. It is more easily removed and does not leave an ulcerated or bleeding surface as in diphtheria. The epithelial layer of the mucosa is rapidly proliferated and enters into



the composition of the pseudomembrane. The mucous membrane is hyperemic and red and in places is denuded of its epithelium.

**Symptoms** — The laryngoscope shows a free fauces, a coated tongue and hyperemia of the fauces and the larynx. The membranous formation appears on the aryepiglottic folds, on the ventricles and occasionally on the vocal cords. It is usually primary in the larynx though it may originate in the fauces and pharynx and spread to the larynx. The temperature rapidly rises to 102° or 103° F.

The onset of the disease may be the same as in acute congestive laryngitis but in the course of an hour or two a loud brassy cough develops which steadily increases. There is loss of appetite and the patient complains of thirst. The pulse is full and the skin is hot and dry. Deglutition becomes painful. The cough at first infrequent becomes more and more frequent and is finally followed by laryngeal spasm. Great dyspnea may ensue and the child in his endeavors to cough out the obstructing membrane clutches at his throat and tosses about in his bed. These symptoms increase in severity as the membrane is formed in the larynx until the voice is aphonic (silent croup) and the inspiration through the narrowed glottis gives rise to a peculiar crowing sound. In case of marked glottic obstruction the inspiratory and expiratory dyspnea and asphyxia may necessitate intubation or tracheotomy.

**Diagnosis** — Membranous croup resembles in some respects spasmodic laryngitis, diphtheria, laryngismus stridulus, retropharyngeal abscess and Vincent's angina of the larynx.

In *spasmodic laryngitis* there is an acute inflammation with spasms of the laryngeal muscles which cause suffocative symptoms. They disappear however in a few minutes and the child rests comfortably. In membranous croup the suffocative symptoms come on gradually and disappear as gradually.

In *diphtheria* the temperature does not rise so high or so rapidly. The chief diagnostic points however are the culture of the Klebs-Loeffler bacilli and the ashen gray and firmly adherent pseudomembrane. After its removal the mucous membrane is ulcerated and bleeding whereas in membranous croup it is smooth and does not bleed.

*Laryngismus stridulus* is a neurosis and not an inflammatory disease hence the laryngoscopic examination shows the absence of inflammation. Then too there is a history of a healthy child who suddenly has a fit of suffocation. In membranous croup there is a history of inflammation and progressive dyspnea.

*Retropharyngeal abscess* may simulate membranous laryngitis in its suffocative symptoms otherwise there is little similarity. An examination of the throat reveals a fluctuating tumor on the posterior wall of the hypopharynx whereas in membranous laryngitis the tumefaction is within the laryngeal zone.

*Vincent's angina* is diagnosed from the smear.

**Prognosis** — Before the advent of the chemotherapy the prognosis was grave. A death rate of from 10 to 50 per cent had been reported.

The prognosis is grave in inverse ratio to the age of the patients. The younger the patient the more serious the prognosis. In adults the danger is greatly diminished as the lumen of the larynx is relatively and actually greater and the mucous membrane is more firmly attached.

**Treatment**—The treatment consists in the early and adequate administration of chemotherapy as indicated by the type of bacteria causing the disease otherwise the treatment is the same as given for acute laryngotracheobronchitis.

If the membrane is due to the spirillum of Vincent and the fusiform bacillus the arsenicals are indicated as in Vincent's angina.

The administration of oxygen or tracheotomy may be necessary if suffocative symptoms develop.

## EDEMA OF THE LARYNX

**Synonym**—Laryngeal edema

Edema of the larynx is an inflammatory process attended with an edematous infiltration of the loose submucous tissue of the larynx which is usually due to a more serious general disease of the heart, kidneys or the liver though it may be caused by local conditions.

**Etiology**—The local causes are mainly traumatic from the injudicious use of caustics, laryngeal injections of irritants, operations, foreign bodies in the supraglottic region of the larynx, the swallowing of hot liquids and the inhalation of hot steam or the inspiration of alcoholic or other irritating liquids into the larynx. It may follow excessive irradiation. Certain drugs such as potassium iodid, ammonia, acetyl salicylic acid etc. may cause it. The prolonged or violent use of the voice as in shouting may bring on edema of the larynx.

Local diseases of the larynx as tuberculosis, syphilis, abscesses, neoplasms, perichondritis, Ludwig's angina and peritonsillitis may also cause it. Abscess of the larynx may be accompanied by a non inflammatory edema.

A non inflammatory edema of the larynx may be secondary to nephritis, diabetes, heart lesions, sclerosis of the liver, angioneurotic edema, myxedema and allergic reactions.

Edema of the larynx may occur in children in association with influenza or the acute exanthemata.

**Pathology**—There is an effusion of clear serum into the laryngeal submucous tissue producing swelling of the aryepiglottic folds and of the anterior and superior parts of the epiglottis. Sometimes the loose subglottic tissue becomes edematous. In associated ulcerative processes the serous infiltration may become seropurulent.

**Symptoms**—The onset is sudden and is characterized by the loss of the voice and rapidly increasing dyspnea. A low pitched stertorous type of breathing is usually present in supraglottic edema (Neffson)<sup>1</sup> in con-

<sup>1</sup> Ann. Otol. Rhinol. and Laryngol. 36: 1065 (December) 1937.

tradistinction to the usual muffled, high pitched stridor of the glottic or subglottic types. In severe cases a fatal issue may occur in from two to three hours by asphyxiation. There is little or no pain or cough.



FIG. 918.—Edema of the larynx. The epiglottis and the arytenoid region show marked swelling.

The laryngoscopic image shows the mucosa in the region of the aryepiglottic folds the anterior and upper surface of the epiglottis and sometimes the subglottic region to be tumefied. The surface of the mucous membrane is a pale gray color in marked contrast to the tumefaction in phlegmonous or inflammatory edema of the larynx in which it is red.

Laryngeal obstruction is recognized by the indrawing at the suprasternal notch the supraclavicular fossæ the intercostal spaces and the epigastrium. An anxious facial expression and restlessness is present. Cyanosis is a late symptom and when present the chances for life are poor.

**Prognosis**—The prognosis may be grave on account of an excessive development of the edema and the serious nature of the constitutional disease back of it. If it is due to an extraneous irritation the danger is less and the chance of recurrence is less.

**Treatment**—If the disease is secondary to a serious constitutional disorder this should of course receive appropriate treatment. Astringent applications of cocaine and adrenaline should be made. Diaphoresis and catharsis should be induced. In addition to the above it may be necessary to puncture the edematous tissue with the laryngeal lancet especially if the edema is non-inflammatory.

Spraying the larynx with a 1 to 100 epinephrine solution may be of great aid in reducing the swelling.

Narcotics are contraindicated if secretions are present due to the danger of abolishing the cough reflex.

If suffocative symptoms appear a low tracheotomy just above the suprasternal notch should be done. The incision may be small that is from 3 to 4 cm. in length. The trachea is incised through the third or fourth ring or even lower.

An intubation tube is not so satisfactory as a tracheotomy because of its tendency to come out its possible irritating qualities to the inflamed and swollen tissues and to the further possibility that the edema may be lower than an intubation tube could reach.

### ABSCESS OF THE LARYNX

**Etiology**—Abscess of the larynx is rare as compared with abscess of the pharynx. It usually follows an acute upper respiratory tract infection. It may be a complication of tuberculous perichondritis. A few

cases follow trauma of the larynx. Typhoid was formerly observed to be the most common cause of this infection. The exact manner in which the abscess forms from typhoid is still debatable. In recent years the majority of the cases reported have followed an acute upper respiratory tract infection usually of the influenza type. Other cases are preceded by scarlet fever, measles, erysipelas, sepsis, gonorrhea and syphilis. The infection may reach the larynx by direct extension or by way of the blood stream. In some cases the infection might be transmitted by way of the lymphatics.

**Pathology** — An osteomyelitis of the thyroid cartilage in adults may be present in which the swelling often remains for a long time. At times a thickening and a fixation of the arytenoid cartilage remains or portions of the cartilages may be sloughed out as sequestra. In perichondritis of the arytenoid the fixation is absolute whereas in the abductor paralysis adduction is still present. In perichondritis of the thyroid cartilage there is a definite tender swelling externally over the cartilage.

In abscesses of the thyroid cartilage swellings of the vocal cords and the ventricular bands, subglottic swellings and occasional fistulae near the commissure occur. Kernan and Schugt found a definite bulging of the outer wall of the pyriform fossa pathognomonic of the exudative process on the lamina of the thyroid cartilage.

Marked swelling of the ventricular bands indicates involvement of the thyroid cartilage whereas swelling of the posterior wall of the larynx beneath the laryngeal aperture speaks more for involvement of the cricoid cartilage (Kernan and Schugt).

The cricoid cartilage is least often affected in abscess formation and perichondritis. When involved a marked swelling of the lamina cricoidea with difficulty in swallowing and usually a swelling and fixation of one or both arytenoids are observed. In adults the process in the cricoid is an osteomyelitis usually unilateral.

**Symptoms** — If the abscess is small a scratching of the throat may be mentioned. If the abscess swelling encroaches upon the glottis there may be loss of voice and intense suffocative symptoms. It is an infectious inflammatory process and causes febrile phenomena. There is retention and pressure hence pain in the larynx. Tenderness on slight pressure over the larynx is present. Small superficial abscesses located on the epiglottis or arytenoids are most frequently seen. They are first observed as small edematous swellings which fluctuate later. The

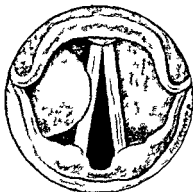


FIG. 1 Abscess of the larynx pointing above the ventricular band.

Laryngoscopic image shows a greatly swollen and reddened mucous membrane at the site of the abscess. Palpation reveals a fluctuating mass. Upon puncturing it with the laryngeal lancet there is a free flow of pus. If the arytenoids are involved swelling and limitation of motion are observed.

If the abscess becomes diffuse marked trismus, high septic temperature, edema and occasionally cyanosis are present.

An extralaryngeal abscess if large may displace the larynx. This may be demonstrated as a rule by the roentgen ray.

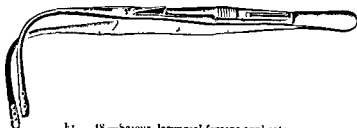


FIG. 18.—Sargous laryngeal forceps applicator.

**Differential Diagnosis**—The condition should be differentiated from retropharyngeal abscess, cysts, new growths, acute thyroiditis, perichondritis, chondritis, etc. If the abscess points to the outside a differential diagnosis should be made from an abscess of a lymph node and an infected cyst of the thyroglossal duct.

**Treatment**—The sulfonamides and the antibiotics should be given as indicated. Spontaneous rupture may occur with complete relief. Hot applications and steam inhalations hasten this result.

Evacuation of the pus by means of a laryngeal lancet may be indicated. This may be done under cocaine anesthesia with the patient in the sitting posture. The anesthesia is induced with a 5 to 10 per cent solution of cocaine applied repeatedly with a laryngeal applicator. The curved laryngeal lancet should then be used with the aid of reflected light and the laryngoscopic mirror or by direct laryngoscopy and the tumor-like mass freely incised. The relief is immediate. If suffocation threatens tracheotomy may be necessary.

If an extensive involvement is present a wide exposure of the larynx through laryngotomy and cricotomy should be done. If the thyroid cartilage alone is involved Kernan and Schugt advocate exposing the cartilage through an external incision, removing the diseased parts and thus draining the abscess. In chronic submucous abscesses of the thyroid cartilage the thyroid cartilage is exposed by means of a window resection of the cartilage exposing the perichondrium. The abscess is then incised and drained. A similar procedure is followed in acute cases with necrosis of the thyroid cartilage.

### HERPES OF THE LARYNX

Herpetic eruptions of the larynx are rare. They are characterized by pain in the throat especially on swallowing and a varying degree of

hoarseness. A few or many vesicles surrounded by a reddish zone may appear especially on the posterior wall of the larynx. The vesicles rupture without scar formation.

The etiology is not known but it probably has the same etiologic factors as herpes elsewhere. Herpes labialis may be associated with herpes laryngis. Acute upper respiratory tract infections seem to be a factor in the initiation of the disease.

The vesicles are round discrete from 1 to 2 mm in diameter and surrounded by a reddish border. They may appear in groups which may become confluent leaving an irregular ulcerated area.

The vesicles are filled with a clear serum which becomes turbid or purulent. The involved areas usually are the posterior laryngeal wall the arytenoid cartilages and the epiglottis. Recovery occurs in from one to two weeks.

The symptoms are mild chills moderate fever a burning and scratching sensation in the throat and later pain on swallowing. A slight hoarseness may be present.

The differential diagnosis should be made from the eruptive fevers such as variola and varicella with vesicles on the mucous membrane of the throat pemphigus with vesicles of the throat and larynx and from a fungus infection with blisters especially the monilia. The prognosis is good. The treatment is symptomatic.

## TRACHEOTOMY

**Surgical Anatomy** — The trachea situated in the exact mid line of the neck is more superficially placed in the laryngeal end than lower down near the suprasternal notch. The muscles overlapping the sides of the trachea leave only a mid line strip of fascia. In the lower portion of the trachea and above the suprasternal notch there is an additional cellular layer containing numerous veins. The thyroid gland on each side of the trachea has an isthmus which crosses the trachea between the third and fourth rings as a rule. From seven to eight tracheal rings are above the suprasternal notch. Important nerves and arteries lie outside the center line of the trachea.

**Indications** — Tracheotomy is indicated when dyspnea or cyanosis follows wounds injuries or stenosis of the larynx or trachea from external strictures swellings obstructions or malformations. It is indicated in rare instances as an entrance for a bronchoscope where peroral endoscopy is impossible. It is frequently used in certain external laryngeal operations such as laryngectomy to maintain an adequate airway or to administer an anesthetic.

Tracheotomy may be done by the high or low operation. The high tracheotomy is made above the isthmus of the thyroid gland which covers the third and fourth tracheal rings as a rule. This high operation has resulted in injuries to the cricoid cartilage with subsequent contractions and other unfortunate sequelæ. It has been abandoned almost universally in favor of the low operation.

**Anesthesia**—Local anesthesia is preferable especially if labored breathing is present. In emergencies time may not permit any anesthesia.

The skin is infiltrated in the exact mid line with novocain epinephrine solution using 10 minims of epinephrine to 1 ounce of a 1 or 2 per cent solution of novocain. The mid line infiltration is made from above the thyroid notch to below the suprasternal notch. The deeper structures require little or no further anesthesia until the trachea is exposed when

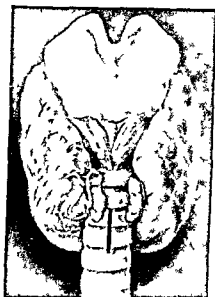


FIG. 213. Tracheotomy. The third and fourth tracheal rings are incised. At times it is necessary to include the second or fifth rings. The isthmus of the thyroid has been divided and tied.

from 10 to 20 drops of a 5 per cent solution of cocaine may be injected (in adults) between the tracheal rings into the interior of the trachea. This tends to reduce the violent cough when the tube is inserted. In infants and children cocaine should be used sparingly or not at all.

**Preparation**—The patient is placed in a supine position on a table with the head extended so as to put the neck on a tension. This is accomplished by placing sandbags under the shoulders. The skin is sterilized from the mandible to below the sternal notch. If the breathing is bad and a bronchoscope is available the preliminary insertion of a suitably sized bronchoscope into the trachea and left in place until the cannula is ready to be inserted simplifies the completion of the tracheotomy. If a bronchoscope is not available a Mosher life saver tube can be inserted into the trachea.

**Incision**—The exact mid line is determined. The trachea is grasped and steadied with the left hand. The skin and fascia are divided in the mid line from the thyroid notch to near the sternal notch.

**Exposure of the Trachea**—The thyroid cartilage should be identified and the inner border of ribbon muscles to each side of the mid line are

separated with the handle of the scalpel and retracted. The pretracheal fascia is incised and separated.

The cleft between the sternohyoid muscles should be identified. The thyroid gland just below and to the sides of this cleft is recognized as a deep pink or red mass protruding between the muscle fibers. The isthmus covering the third and fourth tracheal rings is covered by a fascia. This is divided along its upper border and dissection forceps or the handle of a knife undermines the isthmus from above downwards exposing the bare trachea.

The isthmus is clamped with two pairs of forceps and divided in the mid line. The cut ends of the isthmus are ligated and retracted out of the field of operation. Any excessive venous bleeding should be clamped and tied.

**Incision of the Trachea** —The trachea is fixed with a tenaculum or hook on each side of the mid line. The trachea is now incised in the middle by means of a sharp scalpel through the third and fourth tracheal rings. Occasionally the second and fifth rings are included in the incision depending upon the amount of room necessary for the insertion of the cannula. Care should be taken to avoid cutting the posterior wall of the trachea. Small end segments of one or two of the cut tracheal rings may be severed from each side thereby making a round or oval window in the mid line. If the tracheal rings spread readily this clipping may not be necessary. Mosher uses a punch forceps to make the oval window. When the trachea is first opened a deep breath is usually taken followed by coughing to expel the blood and secretion. This may be followed by a cessation of breathing for a short time but respiration is soon resumed.

**Insertion of the Cannula** —A tracheal dilator or curved forceps is now inserted into the tracheal opening and spread. The tracheal cannula of proper size and length is inserted and held in place by means of tapes attached to the shield of the cannula and tied behind the neck.

A small pad of gauze slit from one edge to the center is placed astride the cannula covering the wound.

**After Care** —A thin pad of fluffed gauze is placed over the opening of the cannula and held in place by a loose bandage so that the airway is not obstructed.

The patient should be kept in a warm well ventilated room with a high moisture content. A well trained nurse should be in constant attendance to remove any obstructing secretions or to attend to other mishaps. The inner tube should be removed and cleansed frequently. A mild suction apparatus by the bed using a catheter of proper size facilitates the removal of the secretion from the tube and trachea.

The outer tube should be changed and cleansed every two or three days if the patient's condition permits. A duplicate tube should be available for insertion if necessary while the first tube is being sterilized. Reinsertion of the tube is greatly facilitated if a pilot is used.

Opiates should be used sparingly if at all due to their inhibiting action on the cough reflex.



**Final Removal of Cannula.**—The cannula may be removed permanently when the normal airway is free and unobstructed. A few days may be sufficient in acute laryngeal inflammation or edema. In some cases of chronic stenosis the cannula may have to be worn permanently. Before removing the cannula it should be plugged temporarily to test the laryngeal breathing.

**Tracheotomy Without the Use of a Cannula**—If an emergency tracheotomy is necessary and a tracheal cannula is not available Mosher's operation may be done.

Mosher (Hill<sup>1</sup>) makes a crucial incision in the trachea, then uses a punch forceps to cut away four triangular flaps. He then sutures the cut ends of the divided thyroid isthmus to the ribbon muscles and they in turn are sutured to the margins of the skin incision. The open wound permits the tracheal window to be near the surface thus obviating the necessity for a tracheal cannula for a few days. All bleeding points must be clamped and tied.

<sup>1</sup> *Ann. Otol., Rhinol. and Laryngol.*, 50, 887, (September) 1941.

## CHAPTER XXVIII

### DIPHTHERIA, INCUBATION

**Definition**—Diphtheria is an acute infectious disease, characterized by the presence of the Klebs-Loeffler bacillus. It is still further characterized by a false membrane on a mucous surface or the abraded skin. It is communicable either directly or indirectly, from one person to another. The lesion is usually located in the upper respiratory tract.

**Etiology**—As to its geographic and racial distribution, it may be said to be well nigh universal. No climate, season, country, or race is exempt from its ravages. It is, however, less prevalent in the summer season in temperate and northern latitudes. Statistics show that among the poor in crowded tenements the disease is more prevalent.

Bodily conditions with lowered resistance have much to do with the susceptibility of the individual exposed to the Klebs-Loeffler bacillus. Abraded or diseased surfaces in the upper respiratory tract also offer local areas of lowered resistance to the growth of the bacilli.

Age has a great influence on the prevalence of the disease. The blood of nurslings is very antitoxic in its properties; hence children under one year of age are comparatively exempt from the disease. After the fourteenth year there is less predisposition to diphtheria.

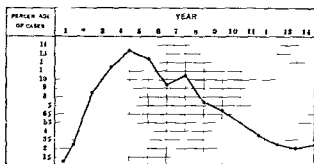


FIG. 250 The above chart is arranged from the statistical data of Babinski and shows at a glance the relative prevalence of diphtheria from birth to fourteen years of age.

**Modes of Infection Direct and Indirect**—The direct infection is from the one affected to another: *i. e.* by breathing or inhaling his breath or receiving the mucus or the saliva into the mouth or the nose during an act of kissing, coughing, spitting or sneezing on the part of the patient. The transmitting media are the discharges from the diphtheritic lesions of the nose, throat, conjunctiva, vagina and wound surfaces and the secretions from the nose and throats of the bacillus carriers.

The indirect mode of infection is not so easily traced as the direct; nevertheless it is suspected that the bacilli may be transmitted by domestic animals as cats, rabbits, etc., which being directly exposed

to the contagion convey it to persons removed from the direct source of infection. It may also be conveyed by towels, table linen and dishes, bedding, books, wall paper, carpets, rugs, clothing and all other articles exposed to a diphtheritic patient. Food, especially milk, may be the source of infection. The hands and the clothing of physicians, nurses and parents should be mentioned as sources of infection.

Diphtheria may be endemic, epidemic or sporadic in its manifestations in a community if proper immunization and quarantine regulations are not carried out.

The distribution of the organism among the human individuals who constitute the sources of infection includes: (a) The frank clinical case, (b) the atypical or missed case, (c) the healthy carrier.

**Carriers** — The atypical or carrier case is the greater problem because these individuals are overlooked and therefore do not come under surveillance.

McGuire and Hitchens published some data on the prevalence of diphtheria carriers in a citizens training camp. Cultures from the throats of 1080 healthy young men between the ages of seventeen and twenty-one years showed that nearly 1 per cent of them carried virulent diphtheria bacilli.

All carriers should have their focus of infection removed if possible or adequately drained if removal is impossible.

It should be a routine practice to remove the tonsils and adenoid from all recovered diphtheritic patients.

**Bacteriology** — The Klebs-Loeffler bacilli vary greatly in size, shape and curvature according to the medium in which they are grown and often vary in the same medium. They also vary with the fluidity, the age and the temperature of the medium, but they generally present the appearance of narrow rods, straight or curved, swollen at either extremity and are found in groups with a tendency to parallelism. They are not always parallel but may have a tangled, irregular arrangement or be in broken chains.

The atypical forms may be thickened at the center of the rod, the extremities being pointed. They may also be lance-, spindle- or club-shaped or even pear shaped. One characteristic is always present, namely, segmentation.

The Klebs-Loeffler bacilli stain readily with alkaline methylene blue and many other aniline dyes.

The diphtheria bacilli may be grown upon blood serum, agar-agar, bouillon, milk, etc., and they are pathogenic for pigeons, rabbits, guinea pigs, chickens, certain small birds, cattle, goats and horses.

**Bacterologic Diagnosis** — A portion of the pseudomembrane should be removed from the throat of the patient with an aseptic cotton wound probe, wire loop or other instrument and smeared over a clean cover glass, dried and stained with Roux's double stain of dahlia violet and methyl green or with Loeffler's blue staining solution.

The cover glass thus prepared should be mounted and examined with a microscope. The diphtheritic bacilli, if present, will be recognized by their typical appearance. If not found, a culture should be made.

which in from twelve to twenty four hours in a temperature of 37° C will develop grayish colonies the size of a pin head with regular outline the surface being dry

Laboratory findings are often nullified by faulty technique in obtaining the culture sometimes it is necessary to dig in in order to get the organism. A negative culture does not always mean absence of diphtheria. A common error is the failure to get a culture from the nose or from the remains of tonsil tissue

**Histopathology**—The distribution of the false membrane may involve the mucous membrane of the nose pharynx tonsils hard and soft palate mouth and lips larynx trachea the bronchi from the largest to the smallest the ear and abraded surfaces of the skin. The vagina the duodenum the conjunctivæ and other mucous membranes may also be involved

Previous to the use of antitoxin autopsies often showed the pseudo membrane extending from the tip of the nose to the smallest bronchi since the use of antitoxin it is rarely found so extensively distributed

The appearance of the pseudomembrane varies from a grayish white through a dirty brown to a black color (in hemorrhagic diphtheria) Its consistency is usually tough and leathery although it may be friable It is firmly attached to the underlying tissues when found on the uvula or the pharyngeal wall and loosely attached in the trachea

The formation of the pseudomembrane begins with an exudation of lymphatic cells which rapidly undergo coagulative necrosis leaving a reticulated substance composed of fibrin from the broken-down cells

If the fibrin penetrates the deeper layers of the mucosa it is difficult to remove it as the line of demarcation is not easily established between the living and the dead tissue. If on the other hand the fibrin remains superficially attached it is easily removed for obvious reasons. When the pseudomembrane is deeply attached its removal is attended by some bleeding if superficially attached there is no bleeding

Sloughing of the mucous membrane may occur when the blood vessels supplying it become thrombosed or otherwise injured so that the nutrition supplied to the parts is shut off. This is often spoken of as gangrenous diphtheria

The appearance in the early stage is usually as a whitish or yellowish circumscribed film and at a still later period it may become yellowish or dirty brown in color. If hemorrhage takes place beneath or within the false membrane it may become black

**General Symptoms** The disease is ushered in by a feeling of discomfort lassitude loss of appetite constipation slight sore throat difficulty in swallowing and more or less hoarseness

The temperature varies with the type however in all types of diphtheria there is an increase of temperature with each extension of the local field of infection. There is a greater fluctuation of the temperature curve in the mixed infection and the septic type than there is in the fibrinous varieties

The pulse rate is increased in uncomplicated cases in the beginning. The pulse-rate in infants is especially high

*Brachycardia* (slowing of the pulse rate) if persistent is a grave symptom

*Tachycardia* (increased pulse-rate) when reaching a rate of 140 or more is a grave symptom. Nasal diphtheria is frequently associated with tachycardia in untreated cases

*Reduced blood pressure* as shown by sphygmographic tracings indicates an increased absorption of diphtheria toxins and warrants a grave prognosis. The same is true of an intermittent pulse

**Diphtheria of the Pharynx** — *Partial angina* is the most common form of the disease. Early there is a general redness of the pharynx and the pillars of the fauces. At the site of pseudomembrane formation which is usually the tonsil there is increased redness. It may form however on the posterior pillars the uvula or the walls of the pharynx. First one tonsil is involved then the other. The cervical lymph nodes are somewhat swollen and tender. The temperature is elevated  $1^{\circ}$  to  $2^{\circ}$  F with frequent oscillations. The general health is good. There is transient albuminuria.

*General angina* is characterized by a thicker and more extensive pseudomembrane gray or dirty yellow in color or even brown or black. The whole or nearly the whole of the tonsils pillars uvula and the pharynx are covered by the membrane in from three to six days if antitoxin is not given. Grave symptoms appear early in untreated cases and are usually ushered in by a chill followed by fever. Delirium restlessness apathy and vomiting are often present. Swallowing becomes difficult on account of the swollen and stiffened condition of the fauces and the pharynx. The nasopharynx is filled with tenacious mucus. The cervical lymph nodes are swollen and tender. Albuminuria is severe. Without treatment the pseudomembrane may be cast off and be reformed continuing thus for three to six weeks. Under antitoxin treatment the disease may be brought under control in from three to six days.

**Septic Diphtheria** — This form of untreated diphtheria in which a secondary infection occurs involves the entire throat from the beginning. The mucous membrane is dark red and the uvula swollen. Within a few hours a dirty gray or blackish membrane forms and rapidly spreads. The cervical lymph nodes are much swollen and very tender. While the membrane is forming and spreading the temperature is elevated. *Toxic symptoms as rapid pulse delirium and restlessness* are present.

As the disease develops vomiting is violent and attended with extreme prostration. The temperature curve rises very suddenly. The pulse is small soft and rapid. Respiration is increased proportionately. The tonsils and the fauces are swollen. They are a livid bluish white with discolored spots. Bloody matter is mixed with the exudate. The cervical lymph nodes are very much swollen and tender on both sides. If untreated death may occur usually on the second to the fourth day from collapse and general sepsis.

**Nasal Diphtheria** — *Diphtheria of the nose* may assume any one of the foregoing types although it is probably more often of the simple fibrous type. It may be primary or secondary. The upper lip is excoriated by

the nasal discharge. The child snuffles, sleeps a great deal and takes food poorly on account of the nasal occlusion and he may become cyanotic in attempting to nurse the breast. The lymph nodes of the neck are swollen. Nasal hemorrhages occasionally take place. Many untreated cases run a benign course while others are malignant from the beginning death occurring within a few days.

The nasal occlusion is at first thought by the parent to be due to a foreign body in the nose. The membrane is usually situated on the septum although it frequently involves the whole Schneiderian membrane and may be removed with the forceps or the syringe as a cast of the interior of the nose.

In the mixed type or *streptodiphtheria* of the nose the symptoms are more severe from the beginning the membrane is mixed with blood and appears black (black diphtheria). Toxic symptoms are marked and the lymph nodes of the neck much swollen and tender. The patients are little inclined to take food. Early antitoxin treatment is usually followed by recovery. The disease is however to be regarded as very grave in its nature if antitoxin is not used.

**Laryngeal Diphtheria (True Croup Membranous Croup Diphtheritic Croup etc.)**—Laryngeal diphtheria may be primary although it is usually secondary to diphtheria of the nose pharynx and tonsils.

**Stage of Invasion**—This is characterized by a simple angina becoming suddenly complicated with hoarseness and a cough characteristic of laryngeal irritation. The Klebs-Loeffler bacillus may or may not be found. A negative finding is not conclusive however as heretofore stated.

**Stage of Spasm (Exudation)**—The pseudomembrane may develop so rapidly that within twenty four hours there is laryngeal stenosis. The cough is dry short and hoarse becoming paroxysmal in character and often lasting for several minutes. It is attended with cyanosis full veins and a perspiring forehead. Aphonia more or less complete soon develops. The respiration is wheezing and noisy. As the stenosis becomes more advanced the inspiratory act is prolonged and is attended with a whistling noise. There is pronounced depression of the supraclavicular region the neck and the epigastrium. The severe symptoms come in waves extreme cyanosis and harsh difficult respiration which gives way temporarily thus affording the sufferer a brief respite from the aggravated symptoms. The natural duration of the stage is from one-half to seven days.

**Stage of Asphyxia**—This stage is characterized by greatly impeded respiration and toxic symptoms. The respiration becomes more rapid and irregular the child sits up suddenly and falls back again exhausted. The cyanosis and the retraction of the supraclavicular jugular and epigastric regions are more pronounced. The suffocative attacks occur more frequently. The head is thrown back and all the accessory muscles of respiration are called into action. Even the abdominal muscles are retracted. The larynx rises with each respiratory effort. During one of the suffocative attacks complicated with convulsions death comes.

**Septic Diphtheria of the Larynx** — This is secondary to a similar process in the nose or the throat or both and begins with fever apathy and marked weakness. The mucous membrane of the larynx and the nose is swollen and covered with a grayish yellow exudate. Toxic symptoms as vomiting delirium suppression of urine heavily coated tongue rapid pulse etc., are marked. The prognosis is quite grave if untreated.

**Diphtheria of the Trachea and the Bronchi** — This is usually secondary to laryngeal diphtheria although it may occur primarily in the bronchi or the trachea. Where it thus forms and the larynx is secondarily involved it is known as ascending croup. If a cast of the bronchi is coughed up it is a positive sign of bronchial involvement. Other signs as respirations (50 to 60 per minute), continuous dyspnea (as contrasted with intermittent when the pseudomembrane is in the larynx and upper trachea) supraclavicular and epigastric depressions not so well marked pale face blue lips and great physical depression may aid in reaching a diagnosis of bronchial diphtheria. The prognosis is very grave.

**Diphtheria of the Ear** — Involvement of the external ear is rare. This is usually carried to the external ear by scratching (abrasion) with the infected fingers of the patient. Infection of the external auditory canal is seen in rare instances in which there is diphtheritic otitis media with extension through the tympanic membrane.

Otitis media as a complication of diphtheria in infants and young children may be quite destructive without perforating the tympanic membrane. However scarlet fever and measles are usually more destructive than diphtheria. The middle-ear infection is characterized by deafness and pain in the ear upon swallowing and coughing these are followed by aural discharge after which the pain subsides.

The purulent discharge may be the only symptom of diphtheria of the middle ear.

The virulency of the culture may be determined by rabbit or guinea pig inoculation.

**Differential Diagnosis** — The differential diagnosis of diphtheria should be made between follicular tonsillitis Vincent's angina agranulocytosis leukemia quinsy retropharyngeal abscess acute laryngo-tracheo-bronchitis tuberculosis and syphilis. The chief diagnostic points in each case are the microscopic and the culture findings.

**Prognosis** — This may be summarized under the following headings.

**The Age of the Patient** — The mortality in untreated cases is the lowest in the first year and the tenth year and the highest in the second to the sixth year of life.

**The Site of the Local Lesion** — Involvement of the larynx if untreated results in the highest mortality. Nasal diphtheria in infants is very fatal if untreated.

Bailey<sup>1</sup> in his series of 5993 cases of diphtheria seen at the Philadelphia Hospital for Contagious Diseases found a death rate from all causes of 8.63 per cent. Excluding those who were hopelessly ill on

admission and who died during the first twenty-four hours in the hospital the death rate was 5.53 per cent.

In the laryngeal cases the death rate for all patients admitted was approximately 25 per cent.

**Complications and Sequelæ of Diphtheria**—The complications of laryngeal diphtheria are dehydration due to lack of fluids, heart lesions, paralysis (peripheral), bronchopneumonia, and other complications such as otitis media, sinusitis, pyelitis, and serum sickness.

**Heart Lesions**—Endocarditis, myocarditis, waxy degeneration, nerve degeneration, heart clots, and dilatation have been found in certain cases. Acute toxic myocarditis is the usual form of heart complication. It usually makes its appearance about seven to fourteen days after the onset of the diphtheria.

**Postdiphtheritic Paralysis**—Postdiphtheritic paralysis has been reported as occurring in from 10 to 20 per cent of untreated cases. The motor nerves are the most often affected, the sensory least. The paralysis usually affects the velum palati (benign and discrete form) and the pharynx. The chief symptom is difficulty in swallowing and the return of liquids through the nose. Each act of swallowing is accompanied by a laryngeal cough. The voice is nasal, articulation is very much interfered with, and the patient snores during sleep. The paralysis disappears in from one to three weeks.

*In the general or diffused postdiphtheritic paralysis* the palatal and the tongue muscles are involved. The muscles of the eye are most frequently affected. Unequal pupils, diplopia, strabismus, or ptosis may be present. Complete recovery eventually takes place. The patellar reflex is impaired or lost, and the muscles of the feet may be paralyzed. The patients shuffle their feet on the floor in walking. Diphtheritic pseudotuberculosis, or even complete paralysis of the lower extremities, may complicate some cases. The muscles of the upper extremities are less often affected. The muscles of the neck and the head are rarely involved. If they are, the child's head falls over on his shoulder. The facial expression may be lost, giving an idiotic cast to the countenance.

Diaphragmatic paralysis occurs in about 7 per cent of untreated cases and may lead to a fatal termination. The chief sign of diaphragmatic paralysis is a sinking in of the abdomen during inspiration, and distention during expiration. Respiration is rapid and panting. Bronchitis or other slight lesion of the lower respiratory tract may lead to asphyxiation and death.

Early and massive serotherapy in the period of angina is the best prophylactic treatment against diphtheritic paralysis, but should be given at any stage of the paralysis if positive cultures are obtained.

**Cardiac or vagus paralysis** complicates about 1 per cent of the untreated cases.

**Bronchopneumonia**—This is a serious complication, and often causes death after tracheotomy and intubation. It is ushered in by a rise of temperature, increased cyanosis (in laryngeal cases), change of the respiration pulse ratio from normal 1.4 to 1.3. At first the physical signs are those of diffuse bronchitis, later of consolidation over several areas.



**Immunization by Antitoxin**—An immunizing dose of antitoxin ranges from 500 to 1500 units according to the age of the patient and the length of time immunity is desired.

Brokaw states the immunity conferred by antitoxin is brief because it is rapidly eliminated from the human body. According to Park 1000 units injected at the time of exposure will give absolute protection to all persons for ten days and to most persons for three weeks.

**Schick Test**—In 1913 Schick published a description of a simple clinical test by which the amount of antitoxin present can be accurately demonstrated. The reaction depends upon the local irritant action of a minute quantity of diphtheria toxin injected intracutaneously. If the individual has no antitoxin or not enough to protect against diphtheria a positive reaction will appear in from twenty-four to forty-eight hours. If the individual possesses antitoxin and is immune to diphtheria, a negative action results.

**Toxin-antitoxin**—In 1913 Behring published the results of his attempts to immunize human beings against diphtheria with neutralized toxin. Three injections of 1 cc each of a suitable toxin-antitoxin mixture spaced one or two weeks apart will cause about 85 per cent of susceptible children or older persons to develop sufficient antitoxin to give the negative Schick reaction and produce marked if not absolute protection against diphtheria. According to Brokaw the toxin-antitoxin injections are inadvisable before the age of six months. During this time most of the infants retain the antitoxin received from their mothers.

**Toxoid**—Diphtheria toxoid is a non-toxic product prepared by treating diphtheria toxin with formaldehyde. It contains no antitoxin or serum proteins. It is used for the active immunization of children against diphtheria.

Diphtheria toxoid is given in two or three subcutaneous injections of 1 cc each four weeks apart. If the patient is still Schick positive after the third injection an additional injection is indicated. Few reactions follow the injections in children under five years of age. In older children and adults a small percentage will develop local and general reactions such as redness, induration, headache and fever.

**Treatment of Diphtheria**—The treatment of diphtheria consists of the administration of adequate doses of antitoxin, certain general and local measures and the relief of the dyspnea by intubation or tracheotomy.

**Antitoxin in Diphtheria**—Introduction of antitoxin in the treatment of diphtheria produced a tremendous reduction in the mortality rate. Brokaw states of 183,000 cases in 150 cities previous to its use the mortality was 38 per cent. Among 132,000 cases after its introduction the mortality was 14 per cent. The time element is most important. No amount of antitoxin can compensate for delayed administration. To wait for the laboratory report may mean a fatality.

The antitoxin may be given in a single dose of 20,000 units intramuscularly unless the case is having much obstruction to respiration, then 2000 units of this are given intravenously.

Schick offers the following suggestions regarding the administration of antitoxin (a) for mild and medium cases 100 units per kilo of body weight (b) for severe cases 500 units per kilo of body weight (c) for immunization 50 units per kilo of body weight, (d) repeated injections should be omitted as superfluous (e) slight improvement in curative results may be obtained by intravenous injection

**General Treatment**—After giving antitoxin the medical care consists in absolute bed rest until all possibility of heart complications has passed. A room or steam tent with a high moisture content and a temperature of 70° F. should be maintained until acute symptoms have subsided. Sedatives may be given as indicated.

**Local Treatment**—The local treatment consists of nasal and oral cleanliness, the removal by suction or swabbing of the excess secretions and membrane. If the pseudomembrane is in the larynx it may be removed by an aspirating tube through a laryngoscope. Removal of this membrane does not leave a bleeding surface as a rule. The membrane may reform in from six to eight hours and require reaspirating a number of times.

### INTUBATION

If the respiratory obstruction is not relieved as determined by increasing depth of the retractions of the intercostal spaces and sternum, increasing pulse-rate to above 160 per minute, pulsus paradoxus, decreasing respiratory rate, increased cyanosis, restlessness and fatigue, an intubation tube should be inserted.

Fschenbrenner<sup>1</sup> in reporting on 600 cases of laryngeal diphtheria believes the first intubation or tracheotomy should be held off as long as possible unless endangering the life of the patient. The first intubation tube is left in seventy-two hours before removal. If the tube is coughed up during that time it is reintroduced if necessary. If the patient cannot get along without the tube after seventy-two hours of intubation he is reintubated for forty-eight more hours.

**Technic of Intubation.**—The child is prepared for intubation by wrapping it in a sheet or a blanket from the shoulders downward. The sheet should be secured with strong safety pins so as to bind the arms and legs of the child. This being done the nurse should sit upright in a chair with the child upon her lap, his head resting against her left breast. His legs should be secured between hers and her right hand should grasp his left and her left hand his right. The assistant should stand behind the nurse and hold the child's head in line with the body with the chin out slightly. A tube of proper size threaded with silk through its eyelet should be in readiness. The operator should stand or sit in front of the child, introduce the mouth gag preferably of the Whitehead type. The operator introduces the index finger of his left hand and hooks it over the epiglottis, locating the space between the two arytenoids and the epi-

<sup>1</sup> Ann. Otol. Rhinol. and Laryngol. 45:480 (June) 1936.

glottis (Fig 202) Then after crowding his finger as far to the left as possible the intubation tube on the introducer is carried into the mouth immediately over the center of the posterior portion of the

tongue the handle of the introducer being on the chest of the child As the tip of the tube passes back of the epiglottis under the finger of the operator the handle should be gradually elevated until the tip of the tube is directly over the chink of the glottis when it should be suddenly lowered thus passing the tube into the box of the larynx and on downward into the glottis and the trachea The tip of the finger

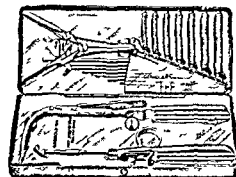


FIG. 201—O'Dwyer's intubation instruments

then engages the rim at the head of the tube the introducer is loosened and removed and with a gentle pressure the tube is firmly pushed deep

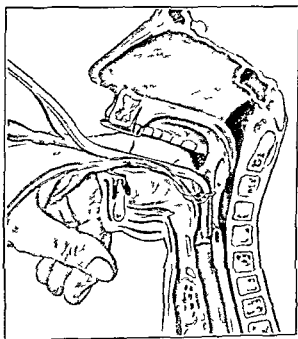


FIG. 202. The index finger holds the epiglottis as the tube passes through the chink of the glottis

into the larynx and the trachea If after waiting twenty to thirty minutes the child tolerates the tube the loop of string should be cut the index finger reintroduced against the head of the tube and the string

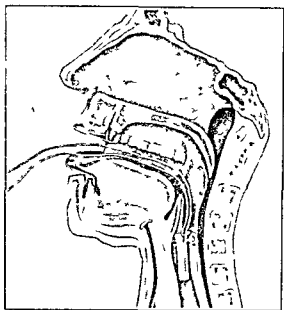


FIG 253 — The tube with thread attached in position in the larynx. The thread may be left for quick withdrawal.

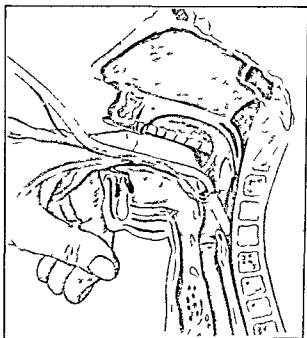


FIG 254 — Making a false passage into the esophagus. The tip of the tube should be introduced by the side of the finger tip and the handle of the obturator elevated until the tube stands perpendicularly, and then passed directly downward through the chuck the glottis.

removed. For obvious reasons the child should be kept wrapped until the string is removed. Figure 254 shows a false entry of the tube into the esophagus because the handle of the introducer was not sufficiently elevated before the tube was dropped into the laryngeal box.

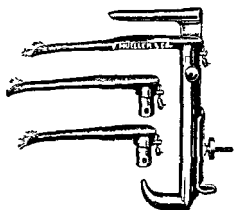


FIG 254—False entry of the tube into the esophagus

Intubation may also be performed in the dorsal position the same relative positions and steps being observed as in the upright position. If preferred intubation may be done by means of Lynch's intubation speculum (Fig 255). This enables the operator to insert the tube by direct vision.

**Extubation or the Removal of the Tube**—The removal of the tube may be done by observing the same precautions as are used in intubation the index finger of the left hand guiding the extractor to the opening

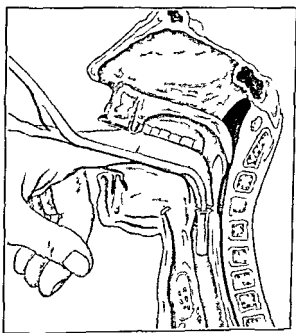


FIG 256—Inserting the extubator for removal of the tube. The finger holds the epiglottis and guides the tip of the extubator into the lumen of the intubation tube.

in the tube (Fig 256). Another method is to leave the silk string attached looping it over the left ear and securing it to the cheek with adhesive plaster. The removal of the tube is thereby rendered quite

easy. It is also easy for the child to remove it hence this is a serious objection to the method. Extubation may be done by bobbing the child wrapped in a sheet is held on a table with its head over the edge face down. Pressure is exerted on the trachea pulling upward as the head is flexed and the tube will fall out on the floor in the majority of cases. The tube may be removed through the laryngoscope under direct vision if desired. One grain of Dover's powder or  $\frac{1}{16}$  to  $\frac{1}{17}$  grain of morphine may be given a few minutes before extubation to prevent spasm and reintubation for its relief. In addition the narcotic reduces the respiratory effort.

**When to Remove the Tube**—Under antitoxin treatment the tube may ordinarily in a child over two years of age be removed in about seventy-two hours. Reintubation may be necessary if the airway is not free. Should the tube become obstructed it should be removed immediately.

**Complications and Difficulties**—(a) If the finger of the operator is short and stubby it may be difficult to introduce the tube beside and beneath it. (b) The tube may make a false passage through the ventricles of the larynx. (c) The prolonged efforts of an awkward or inexperienced operator may cause suffocative symptoms. (d) Transient spasm of the glottis may cause temporary delay in introducing the tube. (e) The narrowest point through which the tube must pass is the cricoid ring and edema or swelling at this point may give rise to some difficulty in introducing it. A smaller one may be passed with slight force. The action of the tube in being expelled in this condition has been aptly said to creep back like an oiled cork in a bottle. (f) Prolonged retention of the tube may be necessary on account of the persistence of the pseudomembrane ulcerations about the cricoid cartilages traumatism cicatricial contractions edema abductor paralysis or exuberant granulations. (g) More rarely the tube may be swallowed (no danger from it). (h) The tube may become obstructed by the thread or catgut being aspirated into it and swollen by the secretions even food may obstruct it. (i) From examination of larynges with the laryngoscope Eschenbrenner<sup>1</sup> found that those cases that need mechanical relief longer than a total of five days usually have a secondary infection of the larynx with other organisms than the diphtheria bacillus. These secondary invading organisms such as the streptococcus staphylococcus and pneumococcus tend to cause much more scar tissue formation than the diphtheria bacillus resulting in a critical stenosis of the larynx. This is particularly true when there is a chronic irritation associated with the infection such as that of an intubation tube in the larynx.

**The Feeding of Intubated Children**—Most cases take liquid food very well when in the upright position although some take it with pain and cough. If the upright position is not practical Casselberry's position may be resorted to. It consists in placing the patient on his back with a pillow beneath the shoulders his head bent downward and backward at an angle of 45 degrees the legs being elevated. Liquid or semi-solid food may be given in this position. The child should be allowed

<sup>1</sup> Ann. Otol., Rhinol. and Laryngol. 45:485 (June) 1936.

to swallow several times before assuming the upright position to remove the food from the nasopharynx. If desired the child may be placed upon its stomach with its head extending over the table and the food drawn upward through a tube. Gavage may be resorted to if the pharynx and the larynx are not too swollen and painful. The tube should be introduced through the nose and rapidly passed into the esophagus. Food being poured into the funnel passes into the esophagus and the stomach. When removing the tube pinch it to prevent the liquid passing into the larynx as it comes out.

Rectal alimentation may be resorted to if feeding by either of the foregoing methods is not practical.

**Permanent Tube Retention**—In some instances it is necessary to leave the intubation tube in place for long periods. These chronic tube cases may be relieved by inserting hard rubber tubes of larger diameter but the same length for from two to four weeks. At the end of this time the tube may be removed in many cases without causing the spasmodic stenosis which would require reintubation. If after two

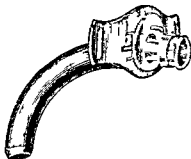


FIG. 257.—Tracheotomy tube

or three days reintubation is not necessary permanent relief is to be expected. If the method fails a low tracheotomy should be done and the intubation tube removed. Laryngeal breathing is restored by gradually plugging the tracheotomy tube. After normal laryngeal breathing has been present for a few days the tracheotomy tube is removed and the wound closed.

**Tracheotomy**<sup>1</sup>—A tracheotomy is seldom necessary especially if the patient is in the hospital and a trained intubator is available. Intubation is usually a safer and surer means of tiding the patient over the suffocative period. Nevertheless there are still cases in which tracheotomy is indicated.

The *indications* for tracheotomy in diphtheria are. When intubation tubes are not available or if for any reason their use is not understood if the pharynx is obstructed from excessive cervical edema and the intubation tube does not give relief when the membrane is in the lower tracheal tract and in the chronic tube cases.

<sup>1</sup> The technique of tracheotomy is described elsewhere.

If the suffocation is not relieved at once after tracheotomy there is either pseudomembrane still lower down in the trachea perhaps a detached piece over the orifice of the cannula or the cannula has become filled with mucus and shreds of pseudomembrane. In this event the inner cannula should be removed and cleared of mucus etc. If the removal of the inner cannula does not relieve the suffocation there is probably membrane low down in the trachea. This may be removed by introducing a catheter attached to a suction apparatus.

The patient should be put to bed face downward with the foot of the bed elevated to facilitate drainage.

The tracheotomy wound is permitted to close from within outward. Gauze may be picked on each side of the trachea to approximate the severed rings as healing progresses. The inner cannula should be removed and cleaned every two or three hours. The outer cannula should be removed and cleaned every twenty-four hours in most instances.

*Mishaps or accidents* which may attend the operation are (a) Failure to open into the trachea especially in very fat children (b) hemorrhage when the incision is carried to either side or too far downward (c) an irregular or too small incision making the introduction of the cannula difficult (d) secondary hemorrhage (e) asphyxiation from dislodged membrane (f) a too greatly retracted head thus flattening the trachea and causing stenosis.

*Complications* which may arise are (a) Infection of the tracheal wound the bronchi and the lungs (b) ulceration of the trachea at the tip of the cannula (c) erysipelas of the wound (d) and most important of all bronchopneumonia from the second to the seventh day after the operation. When this occurs the prognosis is grave.



## CHAPTER XXIX

### CHRONIC DISEASES OF THE LARYNX

CHRONIC inflammation of the mucous membrane of the larynx includes the glandular vascular and connective-tissue layers. It is usually secondary to acute attacks or to overuse or improper use of the voice. It occasionally seems to occur as a primary affection.

The following classification meets both the clinical and the pathologic requirements.

1 Chronic hyperplastic (hypertrophic) laryngitis

(a) Diffused hyperplastic laryngitis sometimes called chronic hyperemic laryngitis

(b) Discrete or localized hyperplasia of the mucous membrane either supraglottic or subglottic

(c) Vocal nodules (chorditis nodosa)

(d) Pachydermia laryngis

2 Atrophic laryngitis

3 Hemorrhagic laryngitis

Each of the four varieties of chronic hyperplastic laryngitis presents a distinct clinical and pathologic picture hence they will be described separately even though they may be in the same general classification.

#### DIFFUSED HYPERPLASTIC LARYNGITIS

**Synonyms**—Hyperemic laryngitis hypertrophic laryngitis sclerotic hyperplasia

This type is characterized by a more or less diffused infiltration throughout the laryngeal mucosa no one part being affected more than another as it is due to irritations of a general character rather than to those directed to one part.

**Etiology**—The exact etiology is unknown. It is extremely doubtful if there is a *primary* chronic laryngitis except from the overuse or improper use of the voice. It is frequently secondary to a preceding disease of the nose nasopharynx or the faucial tonsils. It is possible to have a chronic laryngitis following the excessive use of tobacco or alcohol or even following digestive disturbances. Allergy is a factor in some instances.

Mouth breathing marked nasal obstruction sinusitis polypi overuse of the voice by public speakers and singers may lead to a diffused irritation of the laryngeal mucous membrane. As the improperly prepared air and secretions pass over the whole laryngeal mucosa there may be a diffused inflammation.

Any disease of the heart wherein there is an interference with the return circulation may cause huskiness of the voice and perhaps diffused hyperplasia of the mucous membrane. Thoracic tumors or enlarged

thoracic and cervical lymph nodes, may interfere with the return circulation, and lead to hyperplastic changes

Stone-cutters, tobaccoists, metal-workers and workers with certain chemicals are often affected by chronic laryngitis from the dust laden air

Men are more often affected than women for obvious reasons. The aged are more subject to it on account of the vascular and glandular changes accompanying senility. Orton found the ages of the reported cases ranged from sixteen to fifty-two years

**Pathology**—There is a diffused inflammation and hyperplasia of the laryngeal mucous membrane including the glandular and the connective tissue. The blood vessels are but little affected excepting a few small arteries on the surface of the epiglottis and the vocal cords, where they may be enlarged. Ulcers are absent

Diffused hyperemia of the laryngeal mucous membrane including that of the epiglottis is usually present. It may be more marked in the ventricular pouches, on the epiglottis, the aryepiglottic folds, or on the vocal and the ventricular bands. Indeed it often spreads from one part to another in the order given above until in the later stages it is general. In singers and speakers the hyperemia is generally greater in or is entirely limited to, the true cords. The color varies in different individuals and in the same case at different times. The cords may be the normal ivory-white or pinkish red or they may be streaked with red, or they may be of a pale mottled brown or slate-gray color. One or both cords may show a marked thickening throughout the membranous portion. If both cords are involved the anterior third of the airway may be obstructed (Fig. 258)

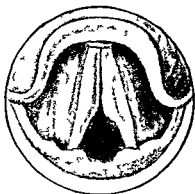


FIG. 258.—Hyperplasia of the anterior portions of both vocal cords

**Symptoms**—The essential symptoms are a husky voice, the sense of accumulated secretions and the ease with which the vocal apparatus becomes tired. The voice upon rising is often quite husky or even aphonic. During the day it becomes nearly or entirely clear unless it is used excessively. In this event it remains husky and its use may be attended with aching in the larynx.

The secretions are increased but little. The image may present either a moist or a dry membrane. The hyperemia is rarely demonstrable by laryngoscopic examination. The mobility of the cords is usually unaffected though in some cases there is a tardy action from the infiltration of the intrinsic muscles.

**Treatment**—The treatment should be addressed to any preexisting etiologic factors if they can be determined.

Complete rest of the vocal apparatus for days or weeks is necessary in many instances. Singers who practise improper placement of the

voice should either be forbidden to sing or be taught proper methods of voice placement. Violent use of the voice either in singing or speaking should be avoided.

The use of sprays and gargles by the patient are of little value. These remedies at most can do no more than thin the secretions and thus facilitate their expulsion.

Excessive hyperplastic growths on the vocal cords may be removed by surgical means.

### LOCALIZED HYPERPLASTIC (HYPERTROPHIC) LARYNGITIS

**Synonyms** — Laryngitis hypoglottica, chondritis vocalis hypertrophica inferior, Stoerk's blennorrhoea.

Discrete or localized hyperplastic laryngitis is characterized by hoarseness or aphonia, dyspnea, a brassy cough, and an infiltration of the tissues in the subglottic space.

**Etiology and Pathology** — The etiology and pathologic changes are the same as those given under the diffuse form, except the pathologic changes are more localized.

Hyperplastic laryngitis frequently occurs in singers due to incorrect methods of voice training and singing, using the voice during or after colds, or from repeated attacks of throat inflammation.

The anterior portions of the cords are usually affected partly or completely, obliterating the anterior third of the airway. At times the interarytenoid space may undergo a hyperplastic enlargement which protrudes into the interarytenoid space like a pad. This hyperplastic mass between the vocal cords prevents their close contact.

**Symptoms** — The symptoms are about the same as those given under the diffuse form, but are greatly exaggerated. The hoarseness occasionally amounts to aphonia. The hyperplastic tissue in the subglottic space and the infiltration of the laryngeal muscles interfere with the normal movements of the cords to such an extent that approximation is often impossible. The dyspnea or suffocative symptoms are due to obstruction below the glottis. The brassy cough is characteristic of obstructive swelling and hyperplasia in the subglottic region.

Examination reveals the hyperplastic masses on or below the cords in the form of two sausage-like masses, nearly parallel with the true cords. Their color varies from a pale grayish pink to the deep red of active inflammation. The epiglottis is also congested and enlarged; blood vessels pass over its posterior surface. In some cases there is more or less edema. In these cases deglutition is difficult, owing to the imperfect closure of the glottis. The dyspnea in discrete hyperplastic laryngitis is increased upon exertion. Patients sometimes complain of a sense of stiffness or of a foreign body in the larynx. After the disease is well advanced the above symptoms are fairly persistent, as the hyperplastic swelling is a fixed factor. Upon attempted phonation the cords fail to approximate, and instead of the free edges presenting straight lines they are slightly concave or wavy, owing to the weakness of the abductor and tensor muscles and infiltration. The secretions are thick and

whitish in color and are often accumulated in the interarytenoid space and over the sluggishly moving cord.

**Differential Diagnosis**—The differential diagnosis should be made from scleroma, laryngeal papilloma, pachydermia laryngis, malignant tumor, tuberculoma, and syphilis.

**Prognosis**—On account of the occasional hyperplastic swellings below the cords, the dyspnea may become so great in rare cases as to require the performance of tracheotomy.

**Treatment**—Before undertaking the treatment, the cause or causes of the affection should be carefully studied. When the etiology has been definitely determined, an endeavor should be made to overcome the predisposing causes of the disease. All nutritional needs such as iron, vitamins, etc., should be met. Any faults in the use of the voice should be corrected. An important part of the treatment is rest of the voice. Iodatum iodide and the protoiodide of mercury should be given whether or not syphilis is suspected, as they often promote more or less absorption of the deposit.

Clerk advocates stripping of the mucosa of the vocal cords on one or both sides as necessary. The affected mucous membrane is grasped and traction exerted along the longitudinal axis of the cord and the hyperplastic mucosa removed. Redundant tissue is removed by straight or bent cupped forceps.

Electrocauterization by means of a pointed laryngeal cautery will cause a shrinkage of the swollen membrane. If a marked hypertrophy is present, excision by the Corde curette or other means is indicated.

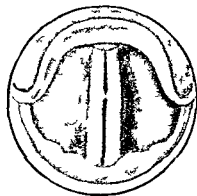


FIG. 209.—Bilateral vocal nodules at the junctions of the anterior and middle thirds of the vocal cords.

## VOCAL NODULES CHORDITIS NODOSA

**Synonyms** Trachoma of the vocal cords, singer's nodules, chondritis tuberosa, pachydermia laryngis.

Vocal nodules are characterized by the formation of nodules along the free border of one or both of the vocal cords at the junction of the anterior and middle thirds.

**Etiology**—The nodules usually complicate chronic hyperplastic laryngitis in singers and public speakers who use faulty methods of respiration and voice placement. When such is the case, there is an overtension of the intrinsic and extrinsic muscles of the larynx. This causes attrition of the cords.

They are seen more frequently in women than in men. Children who habitually shout or scream while playing occasionally develop the nodules.

**Pathology.**—Vocal nodules may be likened to corns due to ill-fitting shoes. Chiari claims that chondritis nodosa is a typical pachydermia laryngis. Hajek thinks the nodules are glandular hypertrophies.

The nodules consist of layers of stratified squamous epithelium surrounded by a circle of congested tissue.

**Symptoms.**—As the nodes frequently accompany a diffuse hyperplastic laryngitis, the symptoms are sometimes similar to those described under that condition. The special symptoms are that the singer or the public speaker is unable to strike the tone he desires, especially in the middle register. When the cords are widely separated, as in the lower register, no difficulty is experienced, as the opposing nodes do not touch. When the higher register is attempted, the posterior thirds of the cords are necessarily closely approximated and not in use, and the voice is not greatly affected. When, however, the middle register is attempted, the cords vibrate their entire length, and as the nodes touch they interfere with voice production.

The laryngoscopic image shows the grayish white nodule on the free border of one or both cords, usually at the junction of the anterior and the middle thirds. If both cords are involved, the nodules are exactly opposite. A small area of hyperemia is often present at the base of the nodule. If diffused hyperplastic changes are present, they may not be apparent except as shown by the hyperemia.

**Prognosis.**—The prognosis in regard to the disappearance of the nodules is good, provided the patient faithfully follows the instructions contained in the chapter on the Singing Voice.

**Treatment.**—The treatment consists in refraining from singing and loud speaking, and in practising proper methods of breathing and tone placement. The patient should be instructed to practise lower costal respiration with the upper ribs elevated, and to practise voice placement by attacking the initial tone with the lips gently closed, as in humming, so that when they are plucked with the finger the tone flows therefrom. If the tone does not emit through the lips when plucked, but comes through the nasal chambers only, it is an evidence of faulty voice placement. If small the nodules may disappear after prolonged voice rest.

If advisable, the astringent remedies described under discrete hyperplastic laryngitis may be used. In extreme cases, it may be necessary to remove the nodules with an intralaryngeal cutting forceps introduced by the direct or indirect method. This should be done only after failure to cure by the other methods suggested. Miffler recommends external massage of the larynx with a mechanical vibrator as an adjunct to proper training in tone building and voice placement. The massage improves the circulation and nutrition of the mucous membrane, increases the local migration of leukocytes, and relieves the associated laryngeal inflammation. Radium or roentgen ray may be of value.

### PACHYDERMIA LARYNGIS

Pachydermia laryngis consists in most instances of a hyperplasia of the squamous epithelium with keratinization.

**Etiology** —The etiology is undetermined but thought to be due to various irritations such as alcohol tobacco irritating fumes or chronic infections of the teeth or sinuses Imperatori<sup>1</sup> believes it is an early form of laryngitis sicca

According to Chiari the verrucous form of pachydermia is identical with papilloma of the larynx and has no relation to the diffuse form Diffuse pachydermia may be primary or it may be secondary to some other affection of the larynx such as tuberculosis or syphilis Typical pachydermia is a very rare disease

**Pathology** —The most frequent and mildest form is a thickening and loosening of the epithelium of the interarytenoid fold and the vocal cords such as frequently occurs in chronic laryngitis Large genuine pachydermia growths in the interarytenoid space interfere very materially with the voice

Circumscribed thickenings outgrowths or nodules which accompany tuberculosis syphilis chronic perichondritis and perhaps also lupus have been referred to as secondary or accessory pachydermia

**Prognosis** —The prognosis depends on the etiology as also does the treatment the latter varying according to the nature of the most distressing symptoms Naturally the syphilitic form is much more favorable than the tuberculous though not infrequently it resists specific remedies

**Treatment** —Operative treatment is called for in suitable cases that is if the general health is good and the respiration or voice is not seriously interfered with by the local disease Unfortunately treatment by means of cutting forceps hot or cold snares etc does not guarantee freedom from recurrence

The method of treatment which is most highly recommended is roentgen ray and radium and the use of electrolysis by means of a bipolar instrument with a current of from 10 to 15 ma The roentgen ray or radium offer the best chances for cure in large pachydermic growths in the interarytenoid space Local applications may be of value Pachydermia laryngitis is often considered to be a precursor of malignancy

## ATROPHIC LARYNGITIS LARYNGITIS SICCA

Atrophic laryngitis is characterized by a burning or pricking sensation after exercising the voice and by suffocative attacks (simulating spasmodic croup and asthma) during the night

**Etiology** —The atrophic changes in the larynx are usually secondary to the same process in the nose and pharynx It is thought that some influence is brought to bear upon the mucous glands of the laryngeal mucous membrane which deprives them of the secretory power and that this influence is often independent of intranasal or pharyngeal atrophy

**Pathology** —The mucous membrane undergoes a retrograde change and fibrous tissue finally replaces the normal elements constituting the

<sup>1</sup> Laryngoscope 41 519 (July) 1931

mucous membrane and submucous tissue. The mucous glands and the blood vessels disappear or become greatly diminished in size. The ciliated columnar epithelium is gradually replaced by squamous epithelium. The secretions are diminished in quantity and changed in quality. They are thicker and admixed with white corpuscles and epithelial debris. The desiccated secretion appears as brownish blackish or grayish crusts on the cords and in the interarytenoid space. Ulceration of the mucosa is not generally present though it may be especially on the posterior wall.

**Symptoms** —After using the voice there may be a burning or pricking sensation in the throat. Cough of a hoarse spasmodic character is excited by the presence of and the attempt to remove the crusts from the larynx. The cough and hoarseness are more severe in the morning. Dyspnea simulating spasmodic croup or asthma may occur at night on account of the accumulation of the crusts over the vocal cords. Upon laryngoscopic examination the mucous membrane appears pale and dry with discolored crusts on the cords or in the interarytenoid space. They may also be seen upon the posterior wall of the larynx in some cases especially if there is ulceration in this region. The cords are dry and wrinkled and more or less covered with crusts. The trachea may be dry and glazed or covered with crusts as well.

**Prognosis** —The prognosis is bad except in those cases in which the atrophic changes have progressed but little.

**Treatment** —The internal administration of the iodids occasionally stimulates glandular activity and thus affords relief. Pilocarpin may also be given for the same purpose if the heart is strong. It should never be given unless an examination of this organ has first been made. The chloride of ammonium stimulates the glands and thins the secretions rendering them easier to dislodge. The inhalation of aromatics in solution in olive oil thrown into the larynx with a nebulizer is grateful and affords temporary relief. Medicated lozenges with a mucilaginous base may be used to protect the dry membrane. A warm moist climate or a sea voyage will ameliorate the symptoms. Careful attention should be given to the condition of the nose the accessory sinuses and the pharynx. If the nose is kept free from crusts and the secretions are increased the larynx will undergo a corresponding improvement. In empyema of the posterior ethmoid and the sphenoid cells the secretions discharge into the pharynx and trickle downward into the larynx where they become dried and adherent to its posterior wall or lodge upon the cords. In such cases great improvement follows the treatment of the sinuses.

### HEMORRHAGIC LARYNGITIS

**Synonyms** —Spurious hemoptysis laryngeal hemorrhage

By hemorrhagic laryngitis is meant a laryngeal inflammation accompanied by hemorrhage from or beneath the laryngeal mucous membrane. The spitting of blood, or hemoptysis is usually not of laryngeal

origin as it may come from the nose pharynx trachea bronchi or lungs

**Etiology**—Hemorrhage beneath the mucosa or otherwise which occurs in the course of laryngitis is due to ulcerations acute inflammations and to excessive use of the voice Syphilis and tuberculosis of the larynx may be attended with laryngeal hemorrhage Albuminuria diabetes hypertension variola typhoid fever yellow fever leukemia hemophilia and malignant disease also predispose to hemorrhages

**Symptoms**—If chronic laryngitis is present the usual symptoms of such a condition are also present The patient also complains of a tickling sensation in the throat followed by cough and the expectoration of blood The quantity of blood varies from a mere streak to a mouthful usually however it is small

The laryngoscopic examination shows one or more areas of extravasated blood in the cords or on or beneath mucous membrane Fresh fluid blood may still cling to the surface of the laryngeal mucosa A hematoma of the cord may be the precursor of a polyp

**Treatment**—Ordinarily no treatment is required Astringent sprays and the external application of ice may be tried If the cough continues it should be quieted by the administration of morphine by hypodermic injection The act of coughing prevents coagulation and tends to prolong the bleeding

**Differential Diagnosis of Chronic Laryngitis** The differential diagnosis of chronic laryngitis from other laryngeal diseases is not always easily made It may be confounded with laryngeal tuberculosis syphilis paralysis carcinoma and certain benign growths

*Tuberculosis* is characterized by a rapid pulse elevation of temperature loss of appetite emaciation a general lowered vitality together with definite lung findings The symptoms are not present in chronic laryngitis An examination of sputum for tubercle bacilli will still further aid in the diagnosis A laryngoscopic examination does not always settle the diagnosis unless the larynx is the seat of the tuberculous infiltration If the tuberculous process is well advanced ulcerations may be present

*Syphilitic* affections of the larynx may present much the same appearance as the edematous type of chronic laryngitis Hyperplasia may be present in both diseases but is more often present in syphilis An accurate history of the case is necessary in making the differential diagnosis In the tertiary stage of syphilis the diagnosis is easily made The ulcers in hyperplastic laryngitis if present are stationary while those of syphilis and tuberculosis are deep and spread rapidly

*Carcinoma* in the subglottic region is distinguished from discrete hyperplastic laryngitis by the nodular or papillary growth of carcinoma and by the biopsy Perichondritis in this region more nearly simulates carcinoma on account of the nodular outline of the tumor like mass

In *lupus* the surface of the membrane is markedly red and granular

*Enchondrosis* of the laryngeal cartilages is differentiated from edema



tous laryngitis by the sense of hardness on probe pressure and the uneven contour of the swelling

*Paralysis* of the posterior crico-arytenoid muscle may be mistaken for subglottic hyperplasia unless a careful examination is made. In paralysis the lagging movements of the cords reveal the nature of the lesion. The paralysis may also be mistaken for pachydermia laryngis.

*Prolapse of the ventricles* is differentiated from hyperplasia by marked pitting upon probe pressure in the former.

*Angioma laryngis* is differentiated from hemorrhagic laryngitis by the elevated whorl of blood vessels and the absence of hemorrhage.

*Papilloma* is distinguished from chondritis nodosa by the point of attachment and the size and shape of the growth.

### DYSPHONIA Plicæ VENTRICULARIS

**Etiology**—Dysphonia plicæ ventricularis or phonation with the ventricular bands as described by C. Jackson and C. L. Jackson<sup>1</sup> is due to underactivity of the true vocal cords from impairment or fatigue of the muscles, impaired innervation or arthritis of the crico-arytenoid joints, overactivity of the ventricular bands possibly due to muscular hypertrophy, absence of the true vocal cords destroyed by disease or following surgical removal, congenital anomaly of the true cords in which the function is absent or impaired, and tumors mechanically propping the cords apart.

**Clinical Anatomy**—The ventricular bands are folds of elastic connective tissue with some fat cells and muscular fibers, the latter from the thyro-arytenoideus. They are in relationship with the aryepiglottic folds externally, the base of the epiglottis anteriorly and the anterior face of the arytenoid posteriorly. The roof of the ventricles is formed by the under surface of the bands. The ventricular bands assist in the closure of the airway and in voice production.

**Symptoms**—The symptoms are those of disturbed phonation. The voice is deep and more or less rough. Double voice (diplophonia, diphthongia) is nearly always present. The voice breaks or two tones are produced at once. A feeling of fatigue in the larynx after using the voice for a prolonged period may be noted. The laryngoscopic appearance resembles inflamed and thickened cords, however the overactive ventricular bands approximate and cover the true cords.

**Diagnosis**—The diagnosis is made from the laryngeal mirror and direct laryngoscopy.

**Treatment**—The treatment consists of the removal of the cause of the dysfunction if possible and the determination of the exact phase of effort of phonation at which the ventricular bands are forced into action. The patient should talk in a low tone with the least possible effort. A prolonged period of silence may be necessary in some cases with chronic inflammatory changes.

<sup>1</sup> Arch. Otolaryngol. 21: 157 (February) 1935.

The Jacksons advise against all irritative forms of treatment. Removing a small bit of tissue from the center of the free edge of each ventricular band may be of value in some instances.

### CONTACT ULCER

Since attention was first called to contact ulcer of the larynx in 1928 (Jackson)<sup>1</sup> 245 cases have been observed.

The ulcer is a superficial one occurring on one or both sides of the larynx posteriorly the ulcerated surface coming in contact on phonation with the same region on the opposite cord the latter being ulcerated or not according to whether the ulceration is unilateral or bilateral (Jackson).

**Etiology**—The disease is nearly always found in adults and as a rule in men. Over use of the voice dust and irritations to the laryngotracheal mucosa may be etiologic factors. Vocal abuse seems to be the chief active etiologic factor. A throaty voice seems to increase the ill effect of the vocal abuse.

The superficial ulcer is usually located in the cartilaginous glottis. Jackson calls attention to the constant hammering of one arytenoid cartilage against the other as the chief mechanical cause. Other etiologic factors present in many cases are suppurative foci in tonsils and sinuses acute infections of the respiratory tract specific infections such as the Vincent's or mixed pyogenic organisms oral sepsis cough tobacco alcohol and chronic laryngitis.

The laryngoscopic appearance usually shows a superficial ulcer of the arytenoid cartilage surrounded by a zone of inflammatory mucosa. The edges of the ulcer are grayish or that of the surrounding mucosa. In some cases a granuloma is seen in the bed of the ulcer.

**Pathology**—The pathologic specimens of tissue show chronic inflammation and superficial ulceration. A thin layer of granulations and necrotic tissue are found in the bed of the ulcer. Granulomas may form in some cases.

**Symptoms**—The chief symptoms are hoarseness and a clearing of the throat. Pain usually is absent but may occur in some cases as a stabbing sensation in the larynx or radiating to the ear. A tickling or a slight stinging sensation may be mentioned. Cough is often present with or without secretions in the larynx.

**Diagnosis**—The diagnosis is made from direct and indirect laryngoscopy and biopsy. The differential diagnosis should be made from the ulcers associated with tuberculosis syphilis malignant and benign growths and pachydermia laryngis. A biopsy may be necessary to establish a diagnosis.

**Treatment**—Complete vocal rest from six months to a year is required. Jackson permits twenty words a day gradually increasing the amount in later months.

Focal infection should be eliminated and oral hygiene instituted. The administration of penicillin by spray and inhalation may help cure any secondary infection present. Silver nitrate is contraindicated. A warm moist climate may be a help.

**Surgical Treatment** — Under direct or indirect laryngoscopy the granuloma is nipped off with cupped forceps flush with the surface. Curettage of the ulcer may be done but in Jackson's opinion is usually less desirable than the use of the cupped forceps. The cautery may be used in treating the granuloma if done in a careful and precise manner. Autogenous vaccines and general supportive treatment may be of value.

### LARYNGEAL ARTHRITIS

The etiologic factors are (a) Secondary to such general infections as typhoid, influenza, syphilis, tuberculosis, etc. (b) it occurs as a concomitant manifestation of involvement of contiguous tissue, as in the cases in which a perichondritic or phlegmonous process makes itself felt in the joints of the larynx, (c) most frequent of all, the condition is encountered as a metastatic focal infection.

The crico-arytenoid joint is affected in the majority of cases. Sometimes the affection of the laryngeal joint is found alone and sometimes it is seen as an accompaniment of a general polyarthritis.

**Symptoms** — The symptoms range from paresthesia of the laryngeal region to pain and hoarseness. The patients may complain of a sensation of fulness or tension in the throat, aggravated by swallowing or speaking.

Crepitation may be elicited by intermittent pressure on the thyroid cartilage. It is characterized by a peculiar grating sensation due to the rubbing together of the inflamed articular lining.

**Treatment** — The treatment is to remove the etiologic factors and such systemic treatment as for arthritis elsewhere.

## CHAPTER XXX

### PARALYSIS AND NEUROSES OF THE LARYNX

Revised and partly rewritten by

JOHN J. BALLINGRIS, B.S., M.D.

The motor nerves of the larynx may be involved in two ways

- 1 Paralysis of the larynx or akinesia i.e. absence of motion
- 2 Spasms of the larynx or hyperkinesia i.e. excessive motion

#### PARALYSIS OF THE LARYNX

**Clinical Anatomy** The intrinsic muscles are supplied by branches of the right and the left vagus (pneumogastric) nerves. These nerves have their origin near the median furrow beneath the floor of the fourth ventricle. Two motor branches the superior laryngeal and the recurrent or inferior laryngeal are given off from each vagus to the larynx.

Sensation to the laryngeal mucous membrane is supplied by the internal branch of the superior laryngeal nerve.

By reference to Figure 260 it will be seen that the external branch of the superior laryngeal nerve supplies only one pair of the intrinsic muscles of the larynx the cricothyroides. These muscles are tensors of the vocal cords hence the wavy outline of the cord (Fig. 261) in superior laryngeal paralysis.

The recurrent or inferior laryngeal nerves supply all the other intrinsic muscles of the larynx namely the arytenoideus the posterior and lateral crico-arytenoids and the internal tensors of the vocal cords.

If the lesion involves all the fibers of the left recurrent laryngeal nerve there is total paralysis of all the muscles of the left side of the larynx except the cricothyroides (external tensor). The same is true of the right side. If the lesion involves only a small branch of the left recurrent one muscle alone may be involved such as the lateral crico-arytenoid. This muscle is an adductor hence there would be incomplete adduction of the anterior two-thirds of the vocal cord on the left side while the opposite cord would slightly encroach beyond the median line. The adduction of the posterior third is controlled by the arytenoideus hence this muscle being unaffected closure in that region is complete. Single muscles are rarely affected except in diphtheria or other local inflammations of the larynx and in small tumors. It is always a question when a single muscle is affected excepting one of the cricothyroid muscles as to whether the lesion is in a nerve twig or in the muscle itself. Inflammatory or neoplastic infiltration may inhibit the nerve twig supplying a certain muscle or the infiltration may cause a mechanical barrier to the proper motion of the muscle. Hysterical paralysis is of course not a true paralysis.

By reference to Figure 261 the course and distribution of the right and the left recurrent laryngeal branches from the vagus is illustrated in diagrammatic form. The left recurrent is given off in front of and at the level of the transverse portion of the arch of the aorta and passes under it thence upward in the groove between the trachea and the esophagus to the muscles of the larynx. As it reaches the larynx it breaks into several twigs thus supplying motor stimulus to all the intrinsic muscles of the left half of the larynx except the cricothyroid

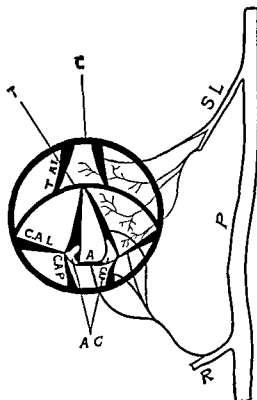


FIG 260—Schema of the nerve supply of the intrinsic muscles of the larynx. *P* the vagus nerve *R* recurrent laryngeal nerve *SL* superior laryngeal nerve *AC* arytenoid cartilages *T* thyroid cartilage *C* cricoid cartilage *A* anterior cricothyroid muscle *C.A.P.* crico-arytenoid posterior muscle *C.A.L.* lateral crico-arytenoid muscle *T.A.I.* internal cricothyroid muscle.

which is supplied by the superior laryngeal. The left recurrent nerve is the one most often affected in paralysis of the larynx on account of its relationship to the arch of the aorta and the left subclavian artery. Aneurysm of the transverse portion of the arch of the aorta causes compression and neuritis of the left recurrent laryngeal and thus inhibits the motor impulses reaching the left half of the larynx. Unilateral paralysis results. Occasionally the aneurysm is so large as to encroach upon the structures on the right side of the chest and may thus also

cause compression of the right recurrent in which event the paralysis would be bilateral

While the right recurrent laryngeal is not so often involved it is nevertheless, so situated with reference to the subclavian artery and the apex of the right lung as to be somewhat frequently the source of laryngeal paralysis. The right recurrent nerve is given off at the level of and

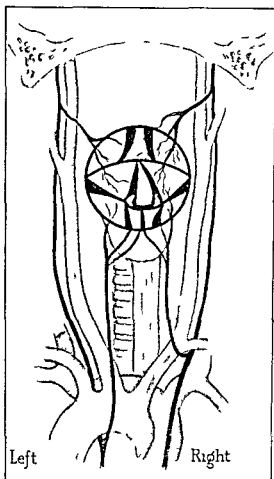


FIG. 261 — Schema (posterior view) showing the relations of the right and left recurrent laryngeal nerves to the vessels of the thorax. Also the distribution of the recurrent laryngeal and superior laryngeal nerves to the intrinsic muscles of the larynx.

anterior to the right subclavian artery and curves below and then behind the latter as it starts upward to the larynx. Aneurysm of the subclavian may therefore compress it and cause laryngeal paralysis of the intrinsic muscles of the right half of the larynx. The right recurrent nerve is in close proximity to the apex of the right lung and may become involved in pleuritic exudates and adhesions in this region, and thus cause paralysis of the right half of the larynx.

**Etiology** — The etiology of paralysis of the vocal cords may be organic or functional. The organic paralysis may be further subdivided into Congenital, central (cortical or bulbar), peripheral, and myopathic.

**Congenital** — Paralysis of the vocal cords of congenital origin is rare. Symptoms are present from birth and the paralysis is usually bilateral.

**Central** — The central lesions responsible for a laryngeal paralysis may be cortical or bulbar. It is generally agreed that a bilateral center for the larynx exists; therefore a unilateral lesion of the cortex would not produce a paralysis of the larynx. In a paralysis of central origin Semon's law may not be true. Kraus in 1884 demonstrated that stimulation of the gyrus prefrontalis in the lower animals produced a contraction, or muscular movements of the larynx, the pharynx and the palate. Semon and Horsley fully substantiated the findings of Kraus by a long series of experiments on the lower animals.

Irritation of one of the external borders of the restiform bodies produces unilateral adduction of the vocal cords. Some cases of unilateral paralysis are also caused by lesions in the medulla or pons, however these are rare.

A bulbar lesion causing laryngeal paralysis usually involves the dorsal motor nucleus of the vagus which lies near the median furrow and is beneath the floor of the fourth ventricle.

In paralysis of the vagus nerve due to a bulbar lesion the involvement of other nerves readily establishes the diagnosis. However an injury to the base of the skull may simulate a bulbar lesion by implicating several nerve trunks in addition to the vagus.

Cases of bilateral paralysis due to bulbar lesions are comparatively rare. The abductors alone are usually involved, however the adductors may be affected in rare cases. The lesions of the bulb that may produce a laryngeal paralysis are progressive bulbar paralysis, syphilis, apoplectic form of bulbar paralysis, stryngomyelia and multiple sclerosis. The paralysis of progressive bulbar palsy usually occurs late and is bilateral as a rule.

In tubes the paralysis is usually of the complete bilateral abductor type but may be unilateral.

**Peripheral.** — Peripheral lesions causing a paralysis of the cords may be located anywhere along the course of the vagus down to and including the recurrent laryngeal. Among the lesions in this locality causing paralysis of the nerves are enlarged cervical lymph nodes, traumatism, goiters (before and following operation), aneurysms, mediastinal tumors, tumors of the esophagus and pharynx, pleurisy, scoliosis of the cervical vertebrae, tuberculosis of the apices of the lungs and even pericarditis or mitral stenosis.

<sup>1</sup> Edinger, *Anatomy of Central Nervous System of Man*. English translation from fifth German edition, p. 375, says:

We have learned then, two nuclei for the vagus, a *ventral one* which from its position (in the prolongation of the ventral horn) and from the appearance of its cells (multipolar with axons passing directly into the nerve) is *motor*, and a *dorsal one* which lies in the prolongation of the gray matter of the base of the posterior horn, is also by its structure characterized as *sensory*.

The left recurrent nerve is probably most frequently involved from an aneurysm of the aorta. The paralysis is usually complete and may be the first and for a long time the only symptom of an aneurysm in this region. If the aneurysm is large a bilateral paralysis may result.

An aneurysm of the innominate or subclavian arteries may produce a paralysis of the right recurrent nerve.

Paralysis of the vocal cords may result from involvement of the vagus nerve at the jugular foramen or from peripheral lesions affecting the superior laryngeal or the recurrent laryngeal nerves in the neck. Lesions of the neck such as injuries, cervical tumors or lymph node involvement, benign or malignant, may be the cause.

Paralysis of the recurrent nerve may occur as a result of disease of the mediastinum or of various pathologic intrathoracic conditions.

Tuberculosis may involve either recurrent nerve. Pathology in the apex of the lung or enlargement of the tracheo-bronchial lymph nodes near the hilum of the lung are liable to involve the nerves. Recovery from this paralysis is the rule.

Syphilis of the larynx may produce paralysis of one or both cords. The paralysis is usually of the complete abductor type, but partial return of function may follow subsidence of the acute process or following treatment. Tracheotomy may be necessary if a bilateral paralysis is present.

In tumors of the nasopharynx the vagus nerve may be involved resulting in paralysis of the vocal cord. The cord is usually in the cadaveric position and involvement nearly always unilateral.

Tumors of the hypopharynx or upper third of the esophagus may produce a paralysis of the vocal cord. In most cases the cord is fixed rather than a true paralysis. This form occurs in men as a rule. It is usually left sided but may be bilateral.

Paralysis of the recurrent laryngeal nerve from pericarditis and mitral stenosis have been observed. It has been attributed to compression or traction. The left cord is usually involved but the paralysis may be bilateral. Recovery may occur if the causative factor subsides.

Toxic neuritis due to various toxic and infectious agents may be responsible such as lead, arsenic, alcohol and atropine or the toxins of diphtheria, influenza or typhoid fever may produce a paralysis usually of the unilateral abductor type.

New and Childrey<sup>1</sup> have observed that a majority of the vocal cords following lesions affecting the recurrent laryngeal nerve are in the median line rather than in the cadaveric position and that the vocal cord returns to its normal position or assumes its normal function within a few months. However, if the lesion causing the paralysis is above the superior laryngeal nerve as may occur from a lesion affecting the vagus at the jugular foramen, the cord is then in the true cadaveric position (New).

The left cord is paralyzed about twice as often as the right. This is probably due to the more central position of the recurrent nerve in the

<sup>1</sup> Arch Otolaryngol 16 143 (August) 1932

<sup>2</sup> Am Jour Med Sci 165 727 (May) 1923



mediastinum New and Childrev found the left cord was more often affected in cases of certain bulbar lesions of tumors of the hypopharynx or esophagus and goiter and even in cases caused by tuberculosis In tuberculosis it would seem that the right nerve would be more frequently involved due to its proximity to the pleura

Davies<sup>1</sup> found an incidence of 0.1 per cent of paralysis of the vocal cord in approximately 8000 cases of benign enlargement of the thyroid gland seen at the Cleveland Clinic This figure is at variance with other reports as an incidence as high as 10 per cent has been reported (Waugh) In the cases of malignant disease of the thyroid about 10 per cent show paralysis of the vocal cord

Tumors traumatism and other lesions at the base of the skull give rise to laryngeal paralysis by implicating the trunk of the vagus It is often difficult to differentiate these conditions from bulbar lesions as they frequently involve the facial glossopharyngeal acoustic spinal accessory, and other branches of the vagus besides the laryngeals depending upon the extent of the lesion

**Myopathic Paralysis**—This form of paralysis is characterized by some local pathologic process in one or more of the intrinsic laryngeal muscles It may be of toxic origin as from typhoid fever or tetanus or it may follow trichinosis tuberculosis local infections and tumors of the vocal cords

### TYPES OF LARYNGEAL PARALYSIS

Paralysis of the larynx may affect either the superior laryngeal or recurrent laryngeal nerves In the former case anesthesia of the larynx would occur in addition to paralysis of the cricothyroid muscles In the latter case any or all of the remaining laryngeal muscles would be paralyzed and the symptoms would depend on what motions were lost to the vocal cords *i. e.*, principally the inability to adduct or abduct the vocal cords

According to their actions on the vocal cords the intrinsic laryngeal muscles may be divided as follows (1) *tensors* cricothyroid muscle and to a less extent thyro-arytenoid muscles (2) *abductors* two posterior crico-arytenoid muscles (3) *adductors* arytenoid muscle lateral crico-arytenoid muscles and thyro-arytenoid muscles, (4) *sphincters* the muscles of the aryepiglottic folds together with the adductor group of muscles (Figs 227 and 228)

**Semon's Law**—Sir Felix Semon<sup>2</sup> and Rosenback<sup>3</sup> have shown that the abductor nerve fibers degenerate earlier than the adductor nerve fibers hence the abductor muscle (mainly the posterior crico-arytenoid) is paralyzed earlier than the adductor (mainly the lateral crico-arytenoid) This phenomenon is usually referred to as Semon's law If therefore the patient is seen early the abductors may be paralyzed If however the patient is examined at a later period the degeneration will have

<sup>1</sup> Arch Otolaryngol 13 435 (March) 1931

<sup>2</sup> Proc Roy Soc London 48 403 1890

<sup>3</sup> Arch f Laryngol u Rhinol 6 588 1897

extended to both the abductor and the adductor nerve fibers and the paralysis will affect both abductor and adductor muscles. This causes the so-called cadaveric position of the vocal cords in which they are midway between complete adduction and the position assumed in quiet respiration.

Semon's law seems to be incorrect so far as it refers to paralysis of central origin; however when applied to peripheral lesions it is essentially correct.

### Functional Paralysis

**Symptoms** In this type of laryngeal paralysis there commonly is a sudden and complete aphonia. Women are much more frequently affected than men and a history of previous attacks can more often than not be gotten. It has its basis in a psychoneurosis usually.

**Diagnosis**—Examination reveals the inability on the part of the patient to adduct the vocal cords, i. e. an adductor paralysis. There is no dyspnea.

**Treatment**—Treatment of the psychoneurosis from which these patients suffer is basic. Any diseased conditions of the body should be eradicated. Some writers have advocated the use of faradization, one pole being placed behind the larynx and the other over the thyroid cartilage. Others advocate a sudden painful lateral displacement of the thyroid cartilage so that the patient verbally protests.

### Unilateral Abductor Paralysis (Unilateral Paralysis of the Recurrent Laryngeal Nerve)

**Etiology**—Unilateral paralysis of the larynx is quite common since each recurrent nerve traverses a long and uninterrupted course before it gives off the terminal twigs to the intrinsic muscles of the larynx. The symptoms are due to the loss of function or paralysis of one of the posterior crico-arytenoid muscles which in turn is usually caused by the paralysis of the corresponding recurrent laryngeal nerve.

**Symptoms**—Hoarseness is the most characteristic symptom. The voice is easily fatigued but there is no dyspnea or cough. Later the unaffected cord compensates for the loss of motion on the affected side and the hoarseness is improved.

**Diagnosis**—The picture seen on examination of the larynx depends on the stage of paralysis as stated in Semon's law. In an early or incomplete stage the paralyzed cord would be seen to lie near the mid line on inspiration. On phonation no abnormality would be found. Later the cord would be fixed in the mid line on inspiration. When the paralysis becomes complete the paralyzed cord would be fixed in the cadaveric position, i. e. midway between the position of phonation and quiet inspiration. Since the tensor (cricothyroid muscle) is not paralyzed the arytenoid on the affected side would lie somewhat anterior to its fellow.

**Prognosis** The prognosis depends upon the cause. If due to a transient inflammation or exudate it is good under appropriate treat-

ment. If due to syphilis the prognosis is good if the case is properly treated. If due to some curable disease the prognosis is correspondingly grave. If dyspnea is present the prognosis is more grave.

**Treatment** — When practical, treat the disease causing the paralysis as in postdiphtheritic or postexanthematic and syphilitic affections. If an incurable disease is carcinoma or sarcoma of the mediastinum or the esophagus is the cause of the paralysis treat the distressing symptoms as they arise.

### BILATERAL ABDUCTOR PARALYSIS BILATERAL PARALYSIS OF THE RECURRENT LARYNGEAL NERVES

**Etiology** — Bilateral paralysis from peripheral neuritis due to diphtheria, typhoid fever, acute infectious diseases and lead poisoning has occurred.

The most common cause of bilateral abductor paralysis is trauma to the recurrent laryngeal nerves at the time of thyroid operations. Laryngeal paralysis due to benign enlargement of the thyroid seems to be very rare.

**Symptoms** — In the early stage the voice is relatively unaffected but inspiratory dyspnea is present at all times. A cough may be present.



FIG. 962. The vocal cords in the quiet respiration.

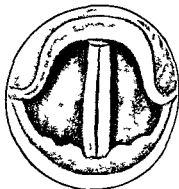


FIG. 963. Complete adduction of the vocal cords as in phonation.

Later when the paralysis is complete the respiration is much easier and dyspnea is present only on exertion. As fibrosis and contraction occur the dyspnea on exertion may increase. The sensibility of the mucous membrane is usually unimpaired unless the lesion of the vagus is above the point where the superior laryngeal nerve is given off. If the recurrent nerve on one side only is affected and the paralysis is complete the vocal cord on that side rests in the cadaveric position while the opposite cord has its normal movements. Indeed it encroaches beyond the median line upon attempted phonation while during deep inspiration it is widely separated from the opposite cord. In one-sided paralysis the position of the arytenoid cartilages is characteristic; the arytenoid cartilage on

the unaffected side overlaps the opposite arytenoid and is either anterior or posterior to it. Cough is usually absent and when present is usually due to an irritation of the trachea by the pressure of a tumor in the neck or upper mediastinum. The cough is like that in aneurysm of the arch of the aorta. Coughing and expectorating are performed with great difficulty in bilateral paralysis.

Dyspnea is absent in unilateral paralysis but may be present in bilateral paralysis in spite of the fact that the cords are separated in the cadaveric position. In the cadaveric position the cords stand midway between quiet inspiration and complete abduction. They are not as widely separated as is usual in inspiration hence the dyspnea.

In some cases the paralysis is partial and the symptoms are therefore correspondingly modified.

**Diagnosis**—In the early stage when the paralysis is still incomplete (*viz* Semon's law) the cords on phonation are seen to approach the mid line fairly well but on inspiration to separate only slightly. In other words the adductor muscles are still working well. Later when the paralysis is complete the cords are always in the cadaveric position whether phonation, inspiration or expiration be tried. By this time both the adductor and abductor muscles are paralyzed.

According to Jackson the terms complete or total paralysis should be used only in the condition of the larynx in which not only are the abductors, tensors and adductors paralyzed but the reflex tonus gone. In this total paralysis the glottic chink is wider and the dyspnea lessened, there is much air waste and the voice is very husky.

**Prognosis**—In view of the serious nature of the causes which produce complete paralysis of one or both recurrent laryngeal nerves the prognosis is grave. In case it is due to syphilitic gummata or to the pressure of enlarged cervical lymph nodes the prognosis under appropriate treatment is good. If due to the toxins of diphtheria or to an acute inflammation complete recovery may occur in a few weeks.

Pulmonary complications may develop from absence of glottic co-operation and the patient may even drown in his own secretions.

**Treatment**—The treatment depends upon the cause of the paralysis and the duration of the symptoms. Any activity predisposing the patient to dyspnea should be avoided. A tracheotomy may be necessary at any time. If enlargement of the thyroid gland is the cause appropriate treatment may diminish the size of the gland and thus relieve the pressure upon the nerve. An operable tumor causing pressure upon the trunk of the vagus or the recurrent laryngeal nerve should be removed in order to relieve the pressure. If the nerve has undergone degenerative changes improvement may be slight or may not result if however the nerve is still healthy the paralysis may disappear after the operation. In aneurysm of the arch of the aorta or of the right subclavian a reduction in size will reduce the pressure on the nerve. Syphilitic gummata may be treated with the various antisyphilitics.

Galvanism and faradism combined with external massage over the laryngeal region may increase the circulation and nutrition of the



FIG. 264 — Calcare position of the vocal cords. The cords are midway between quiet inspiration and complete expiration.

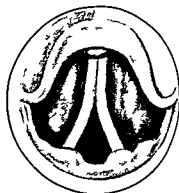


FIG. 265 — Paralysis of the arytenoid muscle.

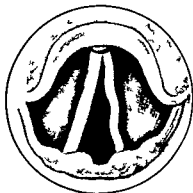


FIG. 266 — Paralysis of the left superior laryngeal nerve with partial atrophy of the cord.

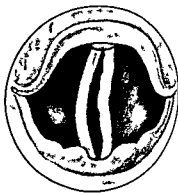


FIG. 267 — Paralysis of the left recurrent laryngeal nerve and external ramus of the right superior laryngeal nerve. During phonation the right cord crosses the midline.

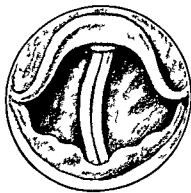


FIG. 268 — Unilateral paralysis of the right recurrent laryngeal nerve. On attempted phonation the normal cord crosses the midline.

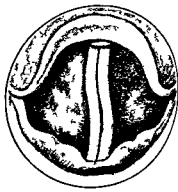


FIG. 269 — Complete bilateral paralysis of the superior laryngeal nerves (cricothyroids) on attempted phonation. The cords are relaxed.

atrophied muscles. Strichnine is also a valuable remedy because it increases the nerve energy and tone of the muscles.

If the paralysis is due to diphtheria or one of the exanthemata the proper treatment should be given to build up the waning and depleted energy. Luminative remedies to stimulate the excretory powers of the intestines, kidneys, liver and skin should be given to clear the toxins from the blood and the lymph.

**Surgical Treatment**—Various methods have been proposed for the surgical relief of this condition. Frazier has suggested an anastomosis of the recurrent laryngeal nerve with the descendens hypoglossi.

Intubation may be performed for the temporary relief of the dyspnea but is not suitable for permanent relief as the tube may be coughed up and its use is uncomfortable to the patient.

Tracheotomy is usually preferable as it affords the least inconvenience to the patient and is ordinarily easily performed. The cyanosis, congestion and edema of the tissues which sometimes complicate the case may however render this procedure difficult to perform. Cordectomy has been tried but seems to offer little or no relief.

McKenry establishes a small permanent opening in the trachea at a point just above where the trachea dips backward into the chest. This gives relief and preserves the voice. The procedure depends on securing a union between the skin and the mucous membrane free from scar tissue enabling the air to pass through the small opening in the trachea in addition to the air inspired through the natural breathing. This counteracts the inspiratory pull on the cords.

**Submucous Resection**—Hoover<sup>1</sup> suggests the submucous resection of the cords and soft tissues of the larynx to widen the lumen of the larynx.

The soft tissue between the mucous membrane and the cartilage of the lateral wall is removed and the mucous membrane placed on the lateral wall. A tracheotomy is done before operation.

The patient is placed on his back, a pad under the shoulders and the head extended on the table. A midline incision is made and carried through the cricothyroid membrane.

One blade of a heavy scissors is introduced through the cricothyroid membrane and carried upward between the cords. The thyroid cartilage and mucous membrane of the larynx are opened in the mid line.

The mucous membrane is separated from the region of the cord and the soft tissue separated from the thyroid cartilage. With a curved scissors or punch the soft tissue and the vocal process of the arytenoid are removed.

The membrane is then sutured along the anterior incision. A small iodoform gauze pack holds the membrane against the cartilage and the end is brought out through the cricothyroid membrane. The cartilage, subcutaneous tissue and skin are brought together and sutured. The tracheotomy tube is left in position. The results have not been entirely satisfactory.

<sup>1</sup> Arch. Otolaryngol. 15: 339 (March) 1929.

### King's Operation

In 1939 Brinn L. King<sup>1</sup> reported an external approach to the arytenoid cartilage in which the crico-arytenoid joint is disarticulated and the arytenoid cartilage displaced outward and held in this position by passing a suture submucously around the arytenoid cartilage<sup>2</sup> and then fastened to the lateral border of the thyroid cartilage. This displacement of the arytenoid cartilage separates the vocal cords sufficiently in most instances to produce an adequate airway. To assist the further opening of the cords during inspiration he attaches the omohyoid muscle to the arytenoid cartilage. However many laryngologists have found transplantation of the omohyoid is not essential to obtain an adequate airway.

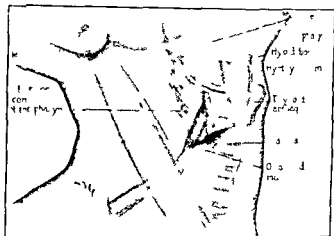


FIG. 20.—The inferior constrictor of the pharynx cut and the superior thyroid artery and vein tied and cut (King, Jour. Am. Med. Assn.)

**Anesthesia**—A preliminary tracheotomy is essential. King uses intravenous pentothal sodium anesthesia but states block anesthesia which includes the internal division of the superior laryngeal nerves would probably be satisfactory.

**Technic—Incision**—An incision 2½ to 3 inches long is made along the anterior border of the sternomastoid muscle passing through the skin and platysma muscle.

**Exposure of the Omohyoid**—The omohyoid muscle is uncovered by separating the tissues between the sternomastoid muscle and the lateral wall of the thyroid cartilage. The inner border and posterior surface of the anterior belly of the omohyoid muscles are freed by dissection. The outer border is not dissected except for about ¾ of an inch at its attachment to the hyoid bone as the descendens hypoglossi nerve and blood vessels are found along this border and injury to them must be avoided.

<sup>1</sup> Jour. Am. Med. Assn. 112:814 (March 4) 1939.

<sup>2</sup> Trans. Am. Assn. for the Study of Goiter 1940.

<sup>3</sup> Trans. Am. Laryngol. Assn. 1941.

The omohyoid is shortened by ligating with chromic catgut from  $\frac{1}{2}$  to  $\frac{3}{4}$  of an inch below the hyoid bone. The excess muscle is cut off distal to the ligature. The cuff created by the ligature enables the sutures to hold when the muscle end is attached to the arytenoid cartilage.

**Exposure of the Arytenoid Cartilage** The fibers of the inferior constrictor muscle of the pharynx is cut where it is attached along the border of the thyroid cartilage. Care must be taken to avoid injury of the internal division of the superior laryngeal nerve just above the tip of the superior cornu of the thyroid cartilage and to the external division of the

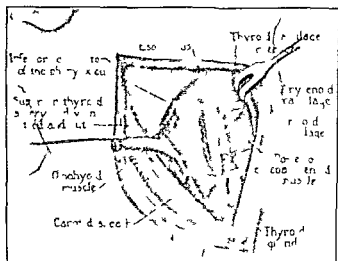


FIG. 1.—The new position of the omohyoid at the close of the operation (Lang J. r. An. Med. Assn.)

same nerve at the inferior cornu. Severing this muscle exposes the pharyngeal mucosa, a delicate structure which is separated from the posterior surface of the cricoid cartilage as far as the midline.

The arytenoid cartilage articulates with the posterior and outer borders of the cricoid. The arytenoid cartilage and the cartilages of Wrisberg and Santorini separate the openings of the larynx and esophagus.

The pharyngeal mucosa is dissected out of the pyriform fossa and away from the posterior spinous border of the arytenoid cartilage. Care must be taken to avoid making an opening into either the pharynx or larynx. This dissection exposes the muscular process of the arytenoid and immediately beneath the muscular process the facet like joint on which the arytenoid sits. This immobilized joint is usually stiff and its ligamentous capsule contracted.

**Mobilization of the Arytenoid**—The muscular process of the arytenoid is identified and a sharp knife inserted under the process into the joint. The joint capsule is then divided by cutting on the mesial outer and posterior sides. Cutting the posterior side divides the fibers of the crico-arytenoideus posterior muscle.

After dividing the capsule the interarytenoideus muscle is cut near



the arytenoid cartilage. Two No. 0 chromic catgut sutures are placed around the mobilized arytenoid, one for displacing the cartilage outward and the second for fastening the severed omohyoid muscle. At times one suture can be used for both purposes.

One suture is passed through a hole drilled or punctured in the wing of the thyroid cartilage. The second suture is then fastened to the cuff end of the severed omohyoid muscle.

Before the sutures are tied a laryngoscope should be inserted to determine the exact amount of airway produced when traction is put on the sutures. If insufficient further mobilization of the arytenoid should be attempted.

For men who do hard labor and in some extreme degrees of contractures King advises cutting a notch in the posterior border of the thyroid cartilage with fixation of the arytenoid into the notch. This increased displacement of the vocal cord would give added airway but would tend to result in a poorer voice.

**Closure of the Wound** — The cut fibers of the inferior constrictor of the pharynx are sutured leaving the omohyoid muscle passing between. The platysma muscle is closed with two or three interrupted catgut sutures. The skin is closed with clips or dermal sutures.

**Comment** — King's experience has led him to the conclusion that it is desirable to try to secure a good result by operating on one side only. The bilateral operation should be used only when a satisfactory result has not been obtained by the first operation. Reoperation on the same side has been unsatisfactory in the few cases he has tried.

The optimum time of operation seems to be after contract on 1 is taken place and not when the cords are in a flaccid state.

### Kelly's Arytenoidectomy

By JOSEPH D. KELLY, M.D.

Arytenoidectomy for bilateral paralysis of the larynx may be performed on either side. However the side on which the cord is most fixed is usually chosen. If both cords are equally immobile the right side is selected.

**Anesthesia** — General or local anesthesia may be used. If local anesthesia is selected the usual preoperative medication is given. The amount and strength of the procaine solution used may be varied with the experience of the operator. Part of the procaine solution used may contain some epinephrine to help control superficial bleeding. Deep injections must be made about the side of the larynx and the hypopharyngeal space as in a laryngectomy operation.

General anesthesia is given through a tracheotomy tube. If the patient is not wearing a tube the tracheotomy operation should be performed under local anesthesia before starting general anesthesia. The operative field is separated from the field of anesthesia by a sterile sheet sewed or clamped to the skin of the neck below the line of incision. In general anesthesia an intratracheal tube (Flagg) may be used to fix the arytenoid cartilage but this is not necessary and if it is used more

care must be exercised to prevent injury and rupture of the intra laryngeal mucous membrane

**Incision and Exposure of the Thyroid Cartilage**—A horizontal incision (Fig 272) is made near the lower border of the thyroid cartilage extending from the median line of the neck to the anterior border of the sterno-cleido-mastoid muscle. The skin platysma myoides muscle and pretracheal fascia are cut and the pretracheal muscles exposed. The sternothyroid sternohyoid and the superior belly of the omohyoid muscles are separated, clamped and cut after the method of a thyroidec-tomy operation. This exposes the thyrohyoid muscle and the wing of the thyroid cartilage. The thyrohyoid muscle is cut and elevated.

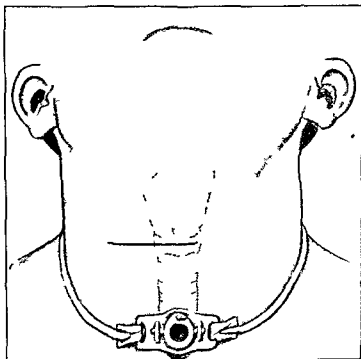


FIG 272—The skin incision for arytenoidectomy (Kelly Arch Otolaryngol)

**Window in the Thyroid Cartilage**—A window (Fig 274) is made in the lower posterior third of the thyroid cartilage below the level of the thyroid notch by means of a sharp pointed knife. The anterior border of the window is limited by a line separating the middle and posterior inferior thirds of the cartilage. The incision is made through the external perichondrium and thyroid cartilage and the cartilage removed with a curette or a small flat nosed rongeur. The size of the window varies with the size of the larynx. A large larynx usually has a large arytenoid cartilage and a small larynx a small arytenoid cartilage. However a window  $\frac{3}{8}$  of an inch square is sufficient and not too large for the ordinary case.

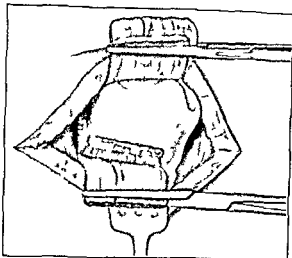


FIG 273 —Exposure of the thyroid cartilage

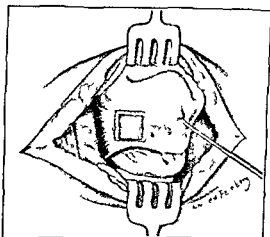


FIG 274 —Exposure of muscle tissue over the arytenoid cartilage (Kelly Arch Otolaryngol)

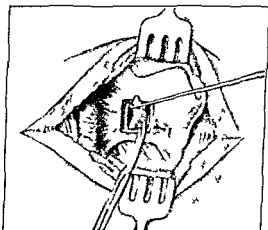


FIG 275 —Dissection of the arytenoid cartilage through the muscle tissue (Kelly Arch Otolaryngol)

**Exposure of the Arytenoid**—The removal of the cartilage brings into view the internal perichondrium of the thyroid cartilage overlying the musculature covering the arytenoid cartilage together with a small branch of the superior thyroid artery and some small veins. The perichondrium is incised with a sharp knife or small scissors and removed. The small branch of the superior thyroid artery is located

about the middle of the upper border of the window. This may be clamped with a small mosquito forceps but pressure with the application of epinephrine usually suffices. By careful dissection with a pair of sharp pointed medium sized plastic scissors through the fibers of the thyro-arytenoid and lateral crico-arytenoid muscles the arytenoid cartilage and the crico-arytenoid articulation are exposed. The capsule of this articulation is severed with scissors and the arytenoid cartilage tumbled from its articulation (Fig 275). With the aid of

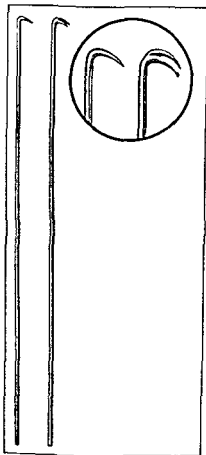


FIG 275—Hooks for lifting the arytenoid cartilage (J D Kelly)

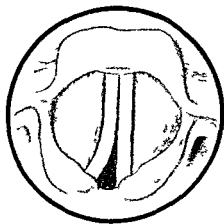


FIG 276—Position of the right vocal cord two months after operation (Kelly Arch Otolaryngol)

hooks (Fig 276) the arytenoid cartilage is lifted and freed of its remaining attachments by careful scissor dissection. The mucous membrane covering the vocal process of the arytenoid cartilage is usually the most adherent and the last tissue to be separated. This area represents the posterior limit of the cord which may be identified by a white line above the muscle tissue. It is at this point that a suture (00 or 000) of mild chromic catgut is placed with a fine ophthalmic needle and the cord pulled away from the median line of the larynx with the suture placed through the internal or external perichondrium.

McCull and Gardner use a brightly lighted anterior commissure laryngoscope to locate the arytenoid from the shadow cast upon the thyroid ala. The window is then made in the region of the shadow.

Orton<sup>1</sup> removes the posterior third of the thyroid ala to obtain a wider field through which to remove the arytenoid. He makes no effort to fix the cord away from the median line. Preservation of as much of the thyroid cartilage as possible would seem advisable.

**Closure of the Wound**—If the intralaryngeal mucous membrane has been ruptured it should be closed with the same suture material and the fibers of the arytenoid muscles approximated to promote healing. The pretrichial muscles are sutured together with mattress sutures using No. 1 or 2 chromic catgut. The platysma myoides muscles may be closed separately with chromic gut and the skin closed with clips.

A cigarette drain is placed under the pretrichial muscles and left in place about five days.

The patients are not decannulated until they can keep the tracheostomy tube corked day and night for two or three days and perform ordinary exertion without discomfort.

## PARALYSIS OF THE SUPERIOR LARYNGEAL NERVE PARALYSIS OF THE EXTERNAL TENSORS OF THE VOCAL CORDS ANESTHESIA OF THE LARYNX

Paralysis of the superior laryngeal nerve is very rare. Childres<sup>2</sup> was able to find only 14 cases in the literature.

**Etiology**—Central lesions which may be responsible for paralysis of the superior laryngeal nerve are locomotor ataxia, paresis, apoplexy, postencephalitic lesions or bulbar lesions. Peripheral lesions may be diphtheria, syphilis, enlarged cervical lymph nodes, injury or neuritis.

**Symptoms**—Anesthesia of the larynx is a prominent and significant symptom. The anesthesia is explained by the fact that it is the superior laryngeal nerve, a branch of the vagus which is affected. This branch supplies the cricothyroid muscles with motor stimulus and the whole of the mucosa with sensation.

A peripheral involvement before giving off the internal and external branches would produce both a sensory and a motor disturbance. The motor paralysis is manifested by a loss of tension of the vocal cord and an inability to control the voice due to the inaction of the cricothyroid muscles. A low pitched voice and inability to sing high tones is characteristic.

When the thyro-epiglottic and the aryteno-epiglottic muscles are paralyzed the epiglottis stands upright, hence the larynx cannot be closed. Because of this and the attending anesthesia food often finds its way into the larynx and upper respiratory tract. No warning is given the patient until the food reaches an area below the vocal cords. Hence pneumonia is frequently a serious sequence. Complete bilateral paralysis of the cricothyroid muscles is manifested by the peculiar way

outlines of the vocal cords (Fig 269). When this paralysis is unilateral the laryngoscope shows one vocal cord on a higher plane than the other.

**Diagnosis**—The peculiar wavy outline of the vocal cord, the local anesthesia, hoarseness and aphonia distinguish it as a true paralysis.

**Prognosis**—It is very bad if there is complete bilateral paralysis but not so very grave when only one cord is implicated. The patient may succumb to inanition or pneumonia. Lobar pneumonia is the usual type and cases have been recorded in which death from this disease could only be ascribed to the passage of food or other foreign substance into the trachea because of the anesthesia. The prognosis is very bad if the recurrent laryngeal nerve is involved at the same time.

**Treatment**—Nourishment by the stomach tube and intravenous solution may be given as indicated. Voice rest is important. The etiologic factors should be eliminated if possible.

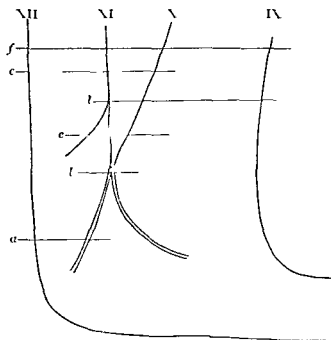


FIG 28—Schema of the IX, X, XI and XII cranial nerves showing the involvement of the nerves in the various syndromes which have been described with a paralysis of the larynx as part of the syndrome. (a) Syndrome of Tappeiner (b) Syndrome of Avellis (c) Syndrome of Schmidt (d) Syndrome of Vernet (jugular foramen syndrome) (e) Syndrome of Jackson (Hughlings-Jackson syndrome) (f) Syndrome of Colet-Scard (after Maurice Vernet)

## LARYNGEAL SYNDROMES

**Syndrome of Avellis**—This syndrome (Fig 278) has been named after Georg Avellis who reported in the *Berliner Klinik* in 1891 a series of illustrative cases giving in detail most of the symptoms of this complex which condition is now recognized by neurologists and laryngologists as an entity.

The syndrome of Avellis may be caused by hemorrhage in the region of the nucleus ambiguus and the spinal fillet in the medulla as well as by cerebrospinal syphilis tuberculosis or syringomyelia. A chronic endarteritis is present as a rule. High blood pressure is a common cause of the hemorrhage.

The syndrome is characterized by an ipsilateral paralysis of the soft palate and vocal cord and partial paralysis of the constrictors of the pharynx and esophagus. A contralateral loss of pain and temperature sense of half of the body below the interauricular line may be present. All other somatic sensations in the areas in which temperature and pain defects occur are retained.

Partial recovery usually occurs.

**Syndrome of Schmidt** — The syndrome of Schmidt is a bulbar syndrome characterized by a paralysis of the vocal cords and palate and a paralysis of the sterno-cleido-mastoid and trapezius muscles. The occiput tilts toward the side of the lesion with the chin to the opposite side.

**Syndrome of Jackson** (Hughlings-Jackson syndrome) — The syndrome of Jackson is a syndrome of the tenth, eleventh and twelfth nerves. It exhibits a paralysis of the vocal cord and of the sterno-cleido-mastoid and trapezius muscles accompanied by a paralysis of one half of the tongue and soft palate.

**Syndrome of Tapia** — The syndrome of Tapia is characterized by an ipsilateral paralysis of the vocal cord, one half of the soft palate and tongue. A swaying and lateropulsion to the right with an ipsilateral loss of sensibility of the side of the face and a loss or a decrease in the pain and temperature sense are present.

**Syndrome of Colet Sicard** — This is a syndrome of the ninth, tenth, eleventh and twelfth cranial nerves. All or part of the symptoms listed in the above syndromes may be present.

## LARYNGEAL PARALYSIS FROM LESIONS OF THE MEDULLA AND THE NUCLEI OF THE SPINAL ACCESSORY NERVE

Laryngeal paralysis from disease or injury of the medulla oblongata and the nuclei of the accessory portion of the spinal accessory nerve is characterized by paralysis of all the intrinsic muscles of the larynx on the side involved or if only a few filaments are involved there will be paralysis of only one or at most two muscles of the larynx. It is still further characterized by the paralysis of certain muscles extrinsic to the larynx which are supplied by nerves having their origin in the immediate vicinity of the motor nucleus of the vagus. Thus there may be paralysis of the facial, the acoustic or of the nerves leading to the extremities.

**Pathology** — Laryngeal paralysis due to a central lesion is dependent upon the involvement of the spinal accessory roots from which some of the fibers of the vagus nerves arise in the floor of the fourth ventricle. There must be a lesion in the medullary or nerve roots supplying the

**larynx.** Syphilis, locomotor ataxia, progressive bulbar paralysis, multiple sclerosis, and tumors of the neck and brain comprise the chief pathologic lesions of central paralysis of the larynx.

**Diagnosis.**—The diagnosis depends on the symptom complex of all the nerves involved. There is usually an associated paralysis of the nerves supplying the tongue, palate, and facial muscles, or of the nerves of audition, or of the extremities. Other regions supplied by the accessory root may be paralyzed. All the intrinsic muscles of the larynx may be paralyzed, or only a part of them, depending on whether all or only a few of the fibers from the vagus motor nucleus are diseased.

**Prognosis.**—The prognosis is nearly always very grave, and even when the disease is due to syphilis it should be guarded, though under antisyphilitic treatment improvement may be expected.

**Treatment.**—The treatment should be varied to meet the symptomatic indications. If syphilis is present, antisyphilitics should be given. If a malignant growth is the cause, treat the unfavorable symptoms as they arise and remove the neoplasm if possible. If marked dyspnea is present from paralysis of the abductors on both sides, tracheotomy should be performed or one of the surgical procedures as given for bilateral abductor paralysis of the recurrent laryngeal nerves may be done.

### SPASMS OF THE LARYNX

Spasms of the larynx may be due to irritation of the central brain cells, whereby all the intrinsic muscles are thrown into violent action, or to irregular nervous impulses sent out from the motor centers of the brain, causing incoordination of the laryngeal muscles.

Paralysis of the intrinsic laryngeal muscles may be limited to one muscle or to a group of muscles, or it may affect all of them.

The spasms may be either tonic or clonic.

Tonic spasms are: of central origin, from irritation of the trunk of the recurrent laryngeal; and from reflex irritation.

**Tonic Spasms of Central Origin.**—In *tubes dorsalis*, spasm of the adductors of the larynx occurs. The clinical picture shows sudden dyspnea with loud inspirations, the cords remaining in adduction for a variable time. It also occurs in tetanus and hydrophobia.

**Tonic Spasm from Irritation to the Trunk of the Recurrent Laryngeal Nerve.**—When the injury is transient and slight, the laryngeal spasm is a forerunner of paralysis. Aneurysm of the arch of the aorta, cancer of the esophagus, pleuritic adhesion of the apex of the right lung, and tumors of the mediastinal lymph nodes may cause the irritation. A slight lesion may also occur in *tubes*.

**Tonic Spasms from Reflex Irritation.**—These may occur from irritation of the larynx, fauces, and neighboring parts. In highly sensitive children irritation in a remote part of the body may cause adduction spasms. The latter condition has been described as laryngospasm infantum, and is usually due to intestinal irritation, tapeworm, tight prepuce, or constipation.



**Clonic Spasms**—Vocal and movements of the larynx and pharynx may be secondary to epidemic encephalitis. These movements are a rhythmic involuntary jerking of the vocal cords soft palate pillars or other musculature of the larynx or pharynx. It is usually accompanied by similar movements in other portions of the face or body.

The muscle relaxes and contracts rapidly so that continuous twitching that varies in speed in different cases is present. The jerking may be universal and may involve most of the voluntary muscles of the body.

Similar movements of the larynx and pharynx have been described in chorea hemorrhage of the brain multiple sclerosis tabes and tumors of the frontal lobe. They are always of central origin.

The condition may last but a few minutes or may persist for many months.

Both tonic and clonic spasms may be present in the same case especially in the depressors of the epiglottis.

Clinically, spasm of the larynx may be classified as follows:

- (a) Spasm of the adductor muscles (laryngismus stridulus)
- (b) Spasm of the tensor muscles
- (c) Spasmodic laryngeal cough or laryngeal chorea

### LARYNGISMUS STRIDULUS (ADDUCTOR SPASM)

**Synonyms**—Spasm of the larynx laryngeal spasm spasm of the adductors of the vocal cords spasm of the glottis spasmus glottidis false croup child-crowing thymic asthma asthma rickets Miller's asthma.

Laryngismus stridulus is a spasmodic act of the intrinsic muscles of the larynx accompanied by stridor. It is a neurosis and is not necessarily associated with laryngeal disease.

**Etiology**—Laryngismus stridulus is often associated with laryngeal or tracheal diseases though it may be a reflex phenomenon from irritation in either contiguous or remote organs. It is sometimes a symptom of acute laryngitis pseudomembranous croup and diphtheritic croup especially in children. It may also occur in non-inflammatory diseases of the larynx. It is common in children but rather rare in adults. It is sometimes associated with intestinal disorders as indigestion worms and constipation. Uterine disorders and sexual excesses have been known to produce it. Disorders of the contiguous organs as the lingual tonsils the teeth (dentition) elongated uvula and inflamed tonsils sometimes excite the spasm. Irritation of the fauces with a brush or a foreign body in the pharynx sometimes causes the symptom. Cases have been reported in which the pressure from an enlarged thymus gland caused laryngismus stridulus. Cerebral irritation caries of the vertebrae and rickets are known causes. Laryngismus stridulus appears in the laryngeal crises of tabes.

**Symptoms**—The outstanding symptom is a sudden laryngeal spasm accompanied by a crowing inspiration usually with the development of cyanosis. The attacks are usually of very short duration relief occurring in from a few seconds to one or two minutes.

**Treatment** — The treatment consists of relieving the source of the irritation rather than applications to the larynx. For the immediate relief from the suffocative spasm the application of cold water to the chest or hot water to the nape of the neck should be made. If suffocation seems imminent and the lower jaw is relaxed seize the tongue between the thumb and the forefinger and exert traction about every three seconds to excite the respiratory center through the reflex action of the phrenic nerve. If the jaw is set the same result can be accomplished by exerting pressure with the fingers under the angles of the jaw.

### APHONIA SPASTICA

**Synonyms** — Spasm of the Tensor Muscles of the Vocal Cords, *Dysphonia Spastica*, *Phonatory Spasms* — Spasm of the tensor muscles is essentially a neurosis from overuse of the voice. The muscles are fatigued and fail to respond to the nervous stimulus sent out from the motor centers of the brain. Writers and telegraphers' cramp are similar affections.

**Symptoms** — Spasm of the tensor muscles is characterized by sudden onset at any moment during speech. It may come on at the beginning or in the midst of a sentence. Patients are seen in whom the speech is suddenly almost or entirely lost for some minutes after which it quickly clears up and remains so for an indefinite period. The patient complains of a rough harsh feeling in the larynx accompanied by the spontaneous flow of a few tears and slight congestion of the conjunctivæ. A drink of water hastens the cessation of the spasms. The cords are tense and approximated in the median line.

**Treatment** — Treatment should be directed to the cause.

In severe and oft recurring spastic aphonia prolonged rest of the voice is necessary. Such cases are usually overtaxed or are affected by a general debility and they should in addition to prolonged rest away from the persons with whom they are daily associated be given tonic or specific remedies to correct the debility or the specific diseases with which each is affected.

*Dysphonia spastica* is a form of hoarseness or a toneless whisper finally resulting in high pitched whistle or screech as the patient continues his effort to speak.

The condition is due to a spasm of the adductors. It usually develops on a neurotic basis in a highly strung adult as a result of vocal strain or shock.

It may have its origin in a pathologic lesion such as laryngitis of a mild nature or from a slight growth of the larynx.

### SPASMODIC COUGH

**Synonyms** — Laryngeal Cough, Laryngeal Chorea, Nervous Cough.

The choreic cough is quite similar to chorea in other parts of the body though it is not usually associated with it. There are however synchronous contractions of other respiratory muscles which furnish the blast of air back of the cough. The choreic cough occurs at frequent intervals and is a dry, noisy, respiratory explosion resembling the velp

or bark of a dog. It occurs most often in females at about the age of puberty, or at the age of greatest instability of the nervous system. It rarely occurs during sleep. Between the intervals the voice is clear. The vocal cords appear normal, and are closely approximated during the attacks.

The nervous cough is a spasmodic, croupy, or even musical laryngeal cough, for which no physical cause can usually be assigned. It is peculiar to neurotic individuals who present other stigmata of a neurosis. It is a "day time" cough, which subsides entirely during sleep, but returns the following morning, often with increased severity. It may be a reflex disturbance from a hypersensitive area in the ear, nose, nasopharynx, or the chest, hence a careful examination of these parts should be made. The sensitive areas in the nose and nasopharynx may be located by gentle probe pressure without the use of cocaine. In the nose Jacobson's tubercle near the anterior end of the middle turbinate may be the seat of the sensitive area. When this is touched with the probe it will give rise to the peculiar nervous cough, provided, of course, that it is the source of the reflex. Impacted cerumen in the external auditory canal may cause it. The reflex may also have its origin in the gastrointestinal tract.

**Treatment.**—The spasmodic cough is due to an hysterical temperament or to a lack of balance of the nervous system at or about the age of puberty, and little can be done to improve it.

As most cases of nervous cough are due to a true neurosis rather than to some physical lesion, the treatment must be of a tonic and sedative character. Antispasmodics and sedatives, as aconite and the bromids, may be given internally to allay the spasms and the local irritation.

### NEUROSES OF THE LARYNX

**Mogiphonia.**—Mogiphonia is characterized by a difficulty in maintaining the tension of the vocal cords while singing, or during forced accentuated speaking. In ordinary conversation no difficulty is experienced.

**Treatment.**—The treatment is rest. Overtaxation being the cause, other forms of treatment are not indicated, unless the condition has recurred often and at frequent intervals. Any etiologic factor should be searched for and removed if possible.

### LARYNGEAL APOPLEXY

**Synonyms.**—Laryngeal vertigo, laryngeal syncope, bronchial syncope, complete glottic spasm in the adult.

Laryngeal apoplexy was first described by Charcot in 1876. It is characterized by a transient irritation and burning sensation in the lower part of the throat, followed by a fit of coughing, dimness of vision, dizziness and unconsciousness, the patient falling to the floor. The disease is a rare neurosis affecting the coordination of the respiratory centers and the nerves of the larynx.

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ratory centers and the nerves of the larynx. The attacks may last but a few seconds, when the spasms cease and the mind becomes clear again. They may recur at intervals of a few weeks.

**Etiology.**—The disease is chiefly found among those leading sedentary lives. Gatchell reported 77 cases ranging in age from seventeen to seventy-seven years. All but 4 were males. Neurasthenia is a rather constant factor but any organic etiologic agent must be ruled out. The possibility of epilepsy and tabes producing the symptoms must be considered.

**Symptoms.**—The face is usually flushed. The disease is clinically like apoplexy with a laryngeal aura and laryngeal spasm, the latter being continued long enough to produce unconsciousness. Such spasms are likely to occur in neurasthenia and in tabes.

**Treatment.**—The treatment should be addressed to the correction of the causative agents.

**Etiology.**—The disease is chiefly found among the well-to-do and those leading sedentary lives. Gatchell reported 77 cases ranging in age from seventeen to seventy-seven years. All but 4 were males. Rheumatism and gout are occasionally associated with it. Neurasthenia is a rather constant factor. Local inflammatory disease of the bronchi, pharynx and larynx is commonly present, and may be an important causative agent.

Among the exciting causes may be named worry from strenuous business or social conditions, and either physical or mental overwork. Dust, smoke, or other irritating substances inhaled into the larynx and bronchi may bring on an attack.

**Symptoms.**—The face is usually flushed, though it may be pale. A deep breath is taken, followed by laryngeal spasm. There may be epileptiform convulsions, and the sequence ends in a few moments by a return to consciousness. After the attack all signs of the disease disappear. The disease is clinically like apoplexy with a laryngeal aura and laryngeal spasm, the latter being continued long enough to produce unconsciousness. Such spasms are likely to occur in neurasthenia and in tabes. Other signs of neurasthenia, epilepsy, and tabes should be sought for before pronouncing the case one of laryngeal apoplexy.

**Treatment.**—The treatment should be addressed to the correction of alimentary and hepatic disorders and to the regulation of the excretory organs of the body. Tonics and antispasmodics may be given to tone and tranquilize the nervous system. Local lesions, if present, should receive appropriate treatment. For instance, bronchitis is the most common concomitant disease, and possibly has something to do with its causation. By relieving the associated diseases of the upper respiratory tract, the laryngeal spasms and the syncope are sometimes entirely relieved.

## CRICOPHARYNGEAL SPASM SPASM OF THE ESOPHAGUS

Cricopharyngeal spasm is a functional stenosis or spasmodic condition of the upper esophagus due to the failure of the cricopharyngeus muscle to relax. This results in the inability of food to pass downward into the

stomach. The cause in many cases is unknown. Foreign bodies or tumors may be the cause in a few cases.

**Symptoms**—Difficulty in swallowing solid foods is a common complaint. Liquids usually pass readily. Pain in the midsternum when swallowing solid or semisolid foods is common.

**Diagnosis**—The roentgen ray usually reveals no pathology other than the difficulty of passing barium through the mouth of the esophagus. As a rule, esophagoscopy reveals nothing other than an exaggeration of the closure of the cricopharyngeus muscle.

**Treatment**—Any etiologic factors found are treated. In the functional cases the passage of a large esophagoscope left in position for a few minutes has been reported favorably (Mainzer<sup>1</sup>). It may have to be repeated a number of times.

### NEURALGIA OF THE LARYNX

True neuralgia is rare and is characterized by pain without a visible cause other than a focal infection. Similar pain may accompany malaria, gout, rheumatism, pressure from some tumor or swelling, nasopharyngitis, and angina of the pharynx.

**Treatment**—The treatment of a neuralgia is to remove the cause. Sedatives may be indicated for temporary relief. Though cocaine if sprayed into the throat affords immediate relief, it is not to be recommended because neuralgic patients easily acquire the cocaine habit. Menthol affords relief. Cold or hot applications to the neck also prove grateful to these patients. Diathermy may be of help.

If the pain is due to a focal infection, malaria, pressure of a tumor or enlarged lymph node, treatment appropriate to these conditions should be instituted.

### PARESTHESIA AND HYPERESTHESIA OF THE LARYNX

Paresthesia or perverted sensations such as 'pins and needles' etc. and hyperesthesia is frequently found in women at the menopause and in neurotic individuals. Organic lesions of the larynx must be carefully excluded.

Treatment should be directed to building up the general health of the patient. Estrogenic therapy may be of distinct value if the menopause is a causative factor.

<sup>1</sup> *Laryngoscope* 45: 386 (May) 1935.

## CHAPTER XXXI

### DEFECTS OF SPEECH

DEFECTS of speech are due to a great variety of causes, most of which are extralaryngeal. The larynx is the primary source of spoken tone, but it is not the complete vocal apparatus. It has been customary in times past to speak of it as the vocal organ, but this can no longer be done in strict conformity to well known facts concerning voice production. While the vibrations of the vocal cords produce the primary tone, it is much modified by the chest, pharynx, nasopharynx, oral and nasal cavities, tongue and the mouth. The character of the tone is also somewhat dependent upon the respiratory system, the best abdominal muscles and diaphragm. The voice changes with age, there is a marked increase in the physiologic activity of the vocal organs as at puberty. This is especially noticeable in the male sex, which exert a marked influence on the quality of the voice. The voice is noted in anger, joy, hatred and love.

It is therefore apparent that defects of speech may arise from parts remote from the laryngeal apparatus. The demands of domestic and social life often make it important that one possess a voice that is pleasing in timbre, range, pitch and modulation, as well as in articulation. Hence attention should be directed to some of the more important lesions which impair the quality and integrity of speech.

Defects of speech may be classified under three heads: (1) defects or faults in tone production (larynx and its attendant resonance cavities); (2) defects or faults in tone formation, modulation or articulation (tongue, teeth, lips, jaws and soft palate); (3) disturbances in speech rhythm (stuttering).

The defects of speech as classified by Travis are: dysarthria, an articulate, labored or ataxic speech due to lesions of the nervous system; uraniscolia, a defect associated with a cleft palate; dyslalia, a hisping mutism or delayed speech; dyslogia, a difficult or incoherent speech usually due to a psychosis; dysphasia, a grouping speech due to a defect in mental imagery; dyschemia, a stammering or stuttering due to a psychoneurosis or emotion; aphonia, a voicelessness; baryphonia, a thick voice; hyperphonia, a whispering; rhinophonia, a nasal voice; dysrhythmia, a defect in rhythm (other than stuttering) usually a defect of rhythm in breathing.

**Defects of Speech of Nasal Origin**—Any occluding lesion of the nose, either of the septum, turbinates or of the nasal fossæ may affect speech, that is they produce those changes in the voice which make it dead, muffled, thick, flat or lacking in resonance.

The speech is still further modified by diffidence, which so often

accompanies complete nasal obstruction. The diffidence, backwardness, or timidity is due to a self-consciousness, to which the defect gives rise. Inability to fix the attention is often attended with diffidence and timidity, and not only is articulation impaired thereby, but fluency and coherency is also somewhat affected.

The elementary sounds of spoken language which depend largely on the resonance of the nasal chambers are not so markedly impaired as those but slightly depending upon it. For instance, the letters *m*, *n*, *b*, and *d* derive their peculiarity from the initial sound, while the final vowel and nasal tones are secondary. Notwithstanding the fact that they are secondary, their absence or suppression makes a noticeable change in the speech, and amounts to a defect. If the final vowel-nasal sound in the above examples were more prominent, the nasal obstruction would not interfere with speech nearly so much, as the speaker could "force" them, and thereby somewhat overcome the apparent effects of the nasal obstruction. The letters *m* and *n* end in a kind of "hum" which is very difficult to produce when nasal obstruction is present, especially when the hum is somewhat suppressed.

The letters *b* and *d* seem to begin with the sound thrown forward against the lips (*b*) and against the tip of the tongue and roof of the mouth (*d*) respectively. The initial sound is, however, made in the larynx and rendered resonant in the chest and nasal chambers. Nasal obstruction modifies the resonance, thus causing a "dead" or "flat" tone to explode at the lips or the tip of the tongue. Thus the speech is rendered defective. We might continue the analysis of the various sounds in speech, showing how nasal obstruction, from one or more of the foregoing conditions, affects the beauty, music, rhythm, and coherency of speech. We might go still farther and show that coherency of thought is impaired also.

**Defects of Speech of Nasopharyngeal and Faucial Origin**—These may be caused by the following: (a) Postnasal adenoid. (b) Fibroma or other neoplasms of the nasopharynx. (c) Chronic hyperplastic changes in the mucosa of the nasopharynx. (d) Marked enlargement of the faucial tonsils. (e) Adhesions of the anterior and posterior pillars of the fauces to the tonsils. (f) Paralysis of the palatine muscles, especially those of the membranous curtain which control the current of air passing to the nares. (g) Paralysis of the soft palate and uvula. (h) Adhesion of the anterior faucial pillars to the base of the tongue. (i) Cleft soft palate and uvula. (j) A shortened soft palate, as is sometimes found after operation for cleft palate.

In the above table the muscular mechanism of speech is affected, and the defects of speech are correspondingly more pronounced than those due to nasal obstruction. The explanation of the more marked defects which seem to have their origin in this classification is not as easy as may appear on first thought. We cannot say that the speech is defective because the muscular action of the parts is interfered with, because many cases come under observation in which there is great muscular im-

pairment but little impediment of speech, while others can scarcely be said to have articulate speech at all; and in still others they cannot be said to have coherent thought. The explanation in some cases is that the muscular impairment existed quite early—before articulate speech was acquired. The impediment thus interfered with the acquirements of articulate speech. The presence of postnasal growths produced mental hebetude (*aprosevia*), heretofore referred to, and the mental ability to acquire articulate speech and consecutive thought was thus impaired. In a few years the growing child becomes more vigorous in mind and body, and makes renewed and voluntary efforts at articulate speech. His failures humiliate and irritate him. He avoids the necessity of speech as much as possible. The speech centers and motor vocal tracts are little used and lie dormant. His mental growth is thereby retarded. The sensitive, reticent child loses the mental growth to be gained by spoken language. He becomes and is regarded as a "backward child."

It becomes the duty and privilege of the rhinologist and laryngologist to loosen the bonds which fetter his imprisoned mind, thus enabling him to enjoy the common pleasures of life, even though he may never become a brilliant member of society.

**Defects of Speech of Lingual Origin.**—The causes may be: (a) Inflammatory adhesions binding the tongue to the anterior faucial pillars and epiglottis. (b) A congenital shortness of the geniohyoglossus muscle. (c) Tongue-tie. (d) Enlargement of the tongue. (e) Excessive enlargement of the lingual tonsils.

Of the foregoing, the most important are adhesions of the tongue to the anterior faucial pillars, tongue-tie, and shortening of the geniohyoglossus muscle. These conditions materially interfere with the articulatory function of the tongue, thus impairing speech. Lipping is a common sign in these conditions. If these lesions exist prior to the acquirement of speech, they may give rise to the clinical picture heretofore referred to under "backward children." The early correction of these physical imperfections may place the child on an equal footing with his fellows.

**Defects of Speech of Laryngeal Origin.**—The etiology may be: (a) Too great strength in the uplifting muscles of the larynx. (b) A weakness of the down-pulling muscles of the larynx. (c) Laryngitis. (d) Chondritis nodosum. (e) Inflammation and infiltration. (f) Perichondritis. (g) Laryngeal arthritis. (h) Mucus or crust accumulations. (i) Neoplasms. (j) Paralysis of the intrinsic laryngeal muscles. (k) Injuries.

If the acute affections of the larynx, as laryngitis, and the chronic conditions, such as chronic laryngeal inflammations and infiltrations, perichondritis, paralysis, arthritis, injuries and neoplasms which cause hoarseness or aphonia, are omitted, there is little to catalogue as causes of defects of speech. This is the more surprising when we recall the fact that the larynx is the primary source of the voice.

Makuen has referred to a condition of the extrinsic muscles of the



larynx which rendered the voice sibilant and *falsetto*. It is given in the table above in *a* and *b*, and is interesting because it illustrates one of the fundamental problems in voice culture, namely, voice placement. If the larynx is allowed to rise too high, the voice becomes *falsetto* and unnatural in quality. If, on the other hand, the laryngeal box is held down in its proper position, the voice assumes its natural register, the tone being pure and pleasing to the ear—that is, it is natural.

The natural and simple things of life appeal most strongly to normal minds. The simple rural scenery, the grandeur of the mountains, the simple melodies of the negroes, the rugged vitality of the Wagnerian opera, and the eloquence of the orator stir the imagination, quicken and fascinate the mind, as the unnatural, the complex, and the artificial cannot do.

Hence, the aim should be to give those having defective speech a speech that is simple and natural. It should be natural in quality, tone, pitch, *timbre*, and rhythm, as well as in modulation and articulation.

**Defects of Speech of Thoracic Origin.**—The causes may be (a) Pulmonary infections such as tuberculosis, pneumonia, etc., or other lesions of the thoracic cavity which may produce the so-called "weak voice" or other modifications. (b) Irregularity of the respiratory rhythm.

Irregularity of the respiratory movements is an almost constant factor in stammerers. Whether this is due to some fault of the respiratory center, or to some peripheral lesion, has not yet been determined. It is probably another manifestation of the unstable nervous system which seems to produce the stammering.

**Defects of Speech Due to Deafness.**—This subject is considered under deaf-mutism, and will only be analyzed briefly here. It may be caused by: (a) Congenital or acquired defect of the auditory apparatus which results in deafness. (b) Nasal and nasopharyngeal diseases. (c) Improper and untimely training or lack of training.

*Congenital defects of the auditory apparatus* are probably present in about one-half of the cases of deaf-mutism, whereas in the balance the defect is due to the ravages of some disease, usually syphilis or the exanthematous fevers. In either instance the child is partially or totally deaf, and cannot, therefore, readily acquire the faculty of speech. He is not mute because the organs of speech are defective, nor because the centers of speech are impaired. Both the peripheral organs of speech and the central mechanism of the brain may be in perfect condition. The child is mute because he cannot hear others speak, and is thereby deprived of the most useful aid in learning—namely, imitation. If he learns to speak, he must be taught by other and more difficult methods. He must be given timely and proper special training. If he has acquired deaf-mutism after having some ability to speak, he may not be a mute in the full sense of the word, but may need some special training to prevent his losing the little speech he already possesses. If the deafness comes before the seventh year of age, there is a strong tendency to lose the faculty of speech, hence, special training is necessary to maintain that already acquired, as well as to broaden it. If the deafness

comes on after the seventh year the patient rarely loses the faculty of speech hence his training can be simpler than that of a child losing his hearing before that age

### STUTTERING—STAMMERING

**Etiology**—The question of whether stuttering is a neurotic or a psychoneurotic manifestation is still undecided. Some believe it is a symptom of the inability of a child to adjust himself to the group caused by fear, timidity or a negative attitude toward the group or that stammering is an oral manifestation of emotional instability and is but one manifestation of emotional maladjustment. Others believe the stutterer cannot inherit stuttering speech but does inherit his neuropathic constitution. Some observers believe that the anxiety and fears of the stutterer are reactions to and are developed after the appearance of his defect.

Speech is of comparatively recent development and requires a complex balanced muscular activity which is easily impaired during periods of intense emotion. The neuromuscular coordination which produces speech is a very complicated one. Conflicting emotions may disrupt this coordination. The autonomic nervous system may be a factor of importance in the mechanics of emotional balance.

According to Elmer I. Kenyon this perversion of the normal speech processes is dependent on emotional disturbance arising from the necessity of developing the speech function under the trying conditions of social interrelations. Thus there are aroused in the child more or less profound manifestations of social emotion. Behind this exciting cause often lie congenital foundation and environmental conditions which tend to encourage the natural childish tendencies to excitability and emotionalism. Natural childish characteristics—impulsiveness, lack of self control, relative absence of knowledge and reasoning, apprehensiveness—as well as the doubtful, wavering, clumsy state of partial development of the speech function, both constitute factors which help to render the child susceptible to this manner of speech perversion.

Kenyon states. The immediate psychology of the incitation to stammering involves (1) emotional excitement, (2) mental confusion and (3) the impulsive effort to talk while in this uncertain state of mind. The result is a speech panic in which normal control of the peripheral speech machine is for the moment lost. In the background of this immediate mental picture lie various disturbing phenomena which add to the mental confusion.

These beginning perversions of the speech act are often repeated the mental stress behind them becomes a more or less constant status of mind. These mental and physical perversions play a baneful part in the general mental and physical physiologic processes of development. Thus the susceptibility to emotionalism and excitability slowly increase both as to uncontrollableness and to intensity and likewise the physical manifestations in the peripheral organs of speech.

This cannot go on for months and years without having its influence in perverting the development of the character in general. The advanced stammerer has become a stammering person rather than an entirely normal person who stammers. This fact renders the complete eradication of the disorder exceedingly difficult and calls for the beginning of treatment at the earliest possible time preferably at the very beginning of the manifestations.

Greene<sup>1</sup> defines the stutter type personality as a chronic hesitation coming from neuropathic stock. The highly excitable nervous system seems to lack the ability to exclude irrelevant stimuli while responding to the normal stimulus. The stutter type belongs to the group of strongly excitable individuals in which their mental and physical activity is disturbed and inhibited because of uncontrolled reactions. His speech is characterized by tonic and clonic spasms of the vocal tract.

Periods of unusual environmental stress may occur first in childhood such as the time the child goes to school or in adolescence. The stuttering frequently begins during these periods.

A probable cause of stuttering has been attributed to a change from left handedness to right handedness on the theory that unchanging neural dominance of handedness from the left to the right side of the brain affects the centers for speech in which confusion often followed by stammering results. The role of sinistrality and ambidexterity as a causative factor in stuttering is unsettled.

About 1 per cent of school children are stutterers. They develop the defect before the age of seven as a rule. Few cases begin in adult life. All stutterers have free periods.

About eight times as many boys as girls stutter probably due to the greater early environmental stress laid on boys.

Physical defects such as enlarged adenoids etc. do not seem to be etiologic factors in stuttering however the correction of such conditions may have a favorable influence upon the stuttering.

**Treatment** — The treatment consists in teaching the conscious control of speech mechanism and at the same time in associating new positive ideas of control, poise and confidence. The patient's obsession that his speech organs will refuse to work, his horror of being the object of ridicule and his dread of being thought inferior—these are the three predominant fears that must be destroyed. Earnest application on the part of the patient and intelligence and understanding on the teacher's side will bring about results in a few months.

Treatment in the form of distraction has been employed extensively. The patient is told to rub or twist a button, swing his arm, play with a watch chain or other object or speak according to tracings etc. In many instances this method is effective.

### PHONASTHENIA—VOCAL FATIGUE

Phonasthenia or functional vocal fatigue results from the improper use of the voice. It may be the result of an improper functioning of any

part of the voice mechanism. It is prone to occur among singers, professional public speakers and those subjecting the voice to unusual strain.

As an indication of improper use of the voice is pointed out by McMahon<sup>1</sup> when the posterior pillars are observed as the patient says 'ah,' they assume a position of an inverted V if improper use of the voice is made rather than an inverted U if the voice is properly used. The voice is too high, loud and hard, particularly with words beginning with vowels. The patient's breathing may be incorrect. Instead of deep breathing, the respiration may be limited to the upper thorax.

A singer may complain that his voice tires easily or that he breaks on certain notes or that a tremolo effect is present. A public speaker may complain of fatigue and lack of clarity of the voice or of hoarseness or lack of voice at times. A scratching, tickling or dryness of the throat may be mentioned.

An improper manner of breathing should be overcome and improper articulation corrected. Talking too loud or too much or singing in an improper range should be corrected.

In a true phonasthenia an inflammatory condition of the larynx is absent. Injection or congestion of the vocal cords sometimes found may be the result of the phonasthenic condition from constant straining or pressing. Laryngoscopic examination usually reveals that upon phonation the cords do not meet in the mid line. This varies from a flaccid condition of the cords to a definite space between them or a difference in level of the cords with an overriding of one arytenoid. According to Bryant the vocal cords under strain are slightly edematous and as the edema subsides the cords do not approximate completely and difficulty ensues. There is an additional psychic element present as a rule.

Massage or light treatments to increase the local blood supply are of great value. Bryant advocates a Faradic current to the region of the thyroid cartilage introduced while the vocal cords are vibrating.

### APHONIA

Aphonia or loss of the voice may be due to acute laryngitis, tuberculosis, syphilis, benign or malignant growths, paralysis of the intrinsic laryngeal nerves following neuritis or pressure on the laryngeal nerves from any cause, hysteria, cerebral lesions affecting the cortical centers governing laryngeal innervation and cerebral concussion.

A functional aphonia may be diagnosed when no known cause is found. A nervous shock may produce it, especially in a neurasthenic patient. Coughing as a rule is not interfered with. The paralysis of the cords is always bilateral.

The aphonia usually disappears spontaneously, occasionally it reappears.

The treatment of this condition is directed to the etiologic factors.

<sup>1</sup> Jour. Laryngol. and Otol. 47: 743 (April) 1937.  
<sup>2</sup> Laryngoscope 43: 607 (August) 1933.

## CHAPTER XXVII

### CHRONIC GRANULOMATA OF THE NOSE, THROAT AND EAR

#### TUBERCULOSIS OF THE LARYNX

**Etiology** — The view that laryngeal tuberculosis is always secondary is held by almost all observers and is proved by the findings of autopsies there being very few recorded cases of death by laryngeal tuberculosis in which either a healed or active pulmonary involvement has not been found.

Tuberculosis of the larynx is found during life in from 3 to 5 per cent of cases of pulmonary tuberculosis but a higher incidence is found on the dissecting table. The incidence of tuberculosis of the larynx is declining. Dworetzky and Risch<sup>1</sup> state the rate has dropped from 20.6 per cent in 1914 to 3.6 per cent in 1941. Laryngeal tuberculosis is very seldom observed before the tenth year. It is most frequent between twenty and forty years of age. Women are most frequently affected during the third decade. Men are somewhat more subject to laryngeal tuberculosis than women. Alcohol and tobacco do not seem to be factors of importance in tuberculosis of the larynx. Other predisposing causes are climate, toxic agents, occupation, local trauma and the general resistance to tuberculous infection whether hereditary, racial or acquired. Pregnancy has a deleterious effect in activating the disease.

The mode of invasion of the larynx is either by direct infection through the inspired air (rare) or by the expectorated sputum (most common) or indirectly by conveyance of bacilli from the tuberculous foci in the lungs through the blood current or lymph channels which is doubtless a less frequent route. Many believe that in cases in which the sputum is free from bacilli the laryngeal involvement is hematogenous. The ventricles and interarytenoid region of the larynx affords a sheltered quiet place for the development of the tubercle bacilli. Supported by a loose connective tissue framework the mucous membrane is thrown into folds by active movement during phonation, respiration, coughing and deglutition. So that infected sputum which is being continually coughed up passes over this irregular surface and much of it remains in constant contact with the deeper crypts especially when the patient is lying down.

With pulmonary tuberculosis established and the expectorated sputum constantly bathing the laryngeal mucous membrane the conditions for infection are quite favorable. The constant presence of the bacilli, the mechanical irritation, the abrasions produced by coughing and the lowered resistance of the cellular structures in general combine to favor such an infection.

**Pathology** — The stages of tuberculosis of the larynx are infiltration, ulceration, perichondritis, and tumor formation. Edema is present in the late stage.

**Infiltration** — In a very early stage type of edema may be absent with only a slight congestion or reddening of the back wall or one of the vocal cord. This is a suspicious symptom of incipient tuberculosis if a pulmonary tuberculosis is present. The ashen gray anemia of the laryngeal mucosa sometimes mentioned is an early sign of laryngeal tuberculosis may be secondary to general anemia. The ashen gray color may be the result of localized edema or fibrotic changes as well.

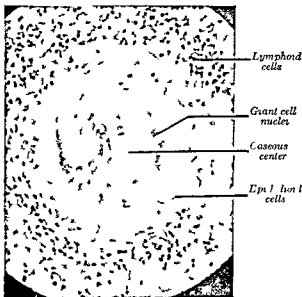


FIG. 9. Large tubercle ( $\times 400$ ). Tuberculosis is a chronic inflammation caused by the tubercle bacillus and the characteristic lesion is the tubercle. In an animal infected for the first time polymorphonuclear cells arrive at the site almost immediately but within twenty-four hours they are replaced by mononuclear cells. These cells represent the essential reaction of the body to the tubercle bacillus. They phagocytize the bacilli and polymorphonuclear cells. After disappearance of the lipid of the bacteria throughout the cell the mononuclears are known as epithelioid cells, which is the single most characteristic feature of the tuberculous reaction. Giant cells are formed by the fusion of a number of epithelioid cells; the nuclei characteristically appear at the periphery. Lymphocytes are seen at the end of a week and form a ring at the periphery of the lesion. By the end of the second week caseation begins in the center of the tubercle. Thus is a form of coagulation necrosis caused by bacterial toxins. The tubercle at this stage presents a homogeneous center, a periphery of pale epithelioid cells with one or more giant cells, and an outer zone of darker lymphocytes. Caseation is not always present.

A somewhat later period of the early stage is that of acute congestion with tubercle formation in which laryngeal symptoms may be absent. The laryngeal congestion may resemble a mild attack of acute congestive laryngitis or the inflammation may center about the interarytenoid region. The infiltration is manifested by redness which may show yellow gray nodules. The yellow nodules or tubercles appear in the

submucosa leaving the epithelium of the mucous membrane intact in the early stage of the infiltration

An early sign is an irregular thickening of the mucous membrane of the posterior wall which may form later a thick pad or mass extending out between the cords. Early involvement of the vocal cords may show a rounding usually of the posterior third giving the cord a spindle shape.



Fig. 780—Tuberculosis of the larynx showing infiltration and swelling of epiglottis, arytenoids and ulcerations of the vocal cords.

The vocal folds are usually infiltrated throughout as the disease progresses. The epiglottis may be puffy the same is the aryepiglottic folds. The arytenoid cartilage may show a swelling at the back wall of the larynx.

**Ulceration**—Through the confluence of separate tubercles large areas develop which loosen the submucosa producing a necrosis of the epithelial coating over the tubercles resulting in a tuberculous ulcer. As a rule the ulcers are shallow at first but deep ulceration may follow. A finely grained caseous matter is found in the bottom of the ulcers.

Incipient ulcerations may be seen by painting with a 2 per cent solution of fluorescein.

The ulcers frequently localize on the squamous epithelium of the vocal cords and the vocal processes. These lesions may spread to the



Fig. 781

Fig. 781—Tuberculosis of the larynx



Fig. 782

Swelling and ulceration of the left arytenoid. Early interarytenoid lesion.

ventricular bands, arytenoids or to the base of the epiglottis. They may spread in rare instances to the mucous membrane of the trachea and large bronchi producing a later bronchial stenosis.

The ventricular bands are not involved as frequently as the cords. When the vocal cords are ulcerated on their free edges they exhibit the typical 'mouse eaten' appearance.

The tendency to ulceration is quite constant. It is rare for a well advanced case of laryngeal tuberculosis to be free from it. The ulcers may be of any size within the limits of the area involved and may be superficial or may extend to the cartilages. They may be discrete or confluent, single or multiple and on one or both sides. When the cartilage is involved by ulceration there is a purulent discharge from the mixed infection. When the arytenoid cartilage is affected the club shaped infiltration tumor is present (Fig. 281). When the infiltration extends to the aryteno-epiglottic ligament the picture is quite characteristic of tuberculosis of the larynx.

The epiglottis is often involved in the process and when infiltrated presents the turban shape so often referred to. The infiltration may extend to both sides of the larynx or be limited to one. When both are affected the view of the deeper portion of the larynx is hidden.

**Perichondritis** — Tuberculous perichondritis is found most frequently on the epiglottis and on the arytenoid cartilages. It is as a rule the result of a deep ulcer in the mucous membrane. In advanced cases softening and liquefaction of portions of the cartilage may take place forming a cartilaginous sequestrum usually on the bottom of the ulcer.

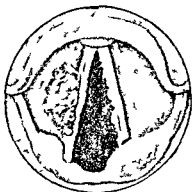


FIG. 283.—Tuberculosis of the larynx. Ulceration of the ventricular band and of the vocal cords.

**Tuberculoma** — A tuberculoma is a tumor like product of tuberculosis which may occur in the form of a fibrotuberculoma or the granulo tuberculoma. The fibrotuberculomas occur usually on the back wall and on the vocal folds. As a rule they are pedunculated. Neither form shows any tendency toward necrosis or caseation.

Pachydermic tumors due to chronic irritation with a secondary hyperplasia of the squamous epithelium are at times seen in tuberculosis.

Slight elevations above the surface due to tubercles or tuberculomas may occur with the infiltration or may follow the infiltrative stage. These may have a smooth exterior but according to Spencer are usually uneven and resemble a papilloma.

Subglottic involvement consists of infiltration with more or less obstruction to breathing.

**Edema** — Edema is nearly always a late symptom. Whenever tubercles are large enough or numerous enough to interfere with the circulation of the larynx edema results. Edema usually occurs on the epiglottis, the aryepiglottic folds, the arytenoid cartilages and the vocal folds.

The different parts of the larynx are usually involved in the following



order (1) the interarytenoid region (2) the arytenoids (3) the vocal cords (4) the ventricular bands (5) the epiglottic and the subglottic regions

Tuberculosis bacilli are found in the sputum in about 90 per cent of all cases of tuberculosis of the larynx. It is usually associated with a productive and exudative cavernous tuberculosis of the lung.

**Symptoms**—Symptoms in the early stages may be absent or the patient may complain of a slight pressure or irritation of the throat or a feeling of dryness and burning. A slight roughness of the voice or frequent clearing of the throat may be mentioned or the voice may tire readily. Hoarseness is a later stage of the disease depending on the spread and the location. Even a slight involvement in one or both vocal cords may cause no noticeable hoarseness. The hoarseness may be intermittent or only noticed toward the evening.

As the laryngeal involvement is usually secondary to the pulmonary there is more or less cough, often without expectoration.

Pain on swallowing develops if the epiglottis, arytenoid cartilages or the aryepiglottic folds are involved. The pain is most severe if a bilateral involvement is present.

In the stage of infiltration and ulceration the hoarseness, cough and dysphagia become prominent symptoms.

In the stage of perichondritis and necrosis all symptoms are exaggerated. The voice may be completely gone, the cough incessant and the pain marked. Death from the pulmonary involvement may occur before this stage is reached.

In the late stage dyspnea may be present. It is in proportion to the degree of edema, infiltration and the cicatricial contraction.

In examining the larynx two methods are in use, the direct method with illuminated instruments and the indirect method with the laryngeal mirror. The direct method by lifting the epiglottis gives an excellent view of the whole interior of the larynx.

**Differential Diagnosis**—Laryngeal tuberculosis must be differentiated from syphilis, carcinoma, lupus, chronic laryngitis, scleroma and actinomycosis.

Syphilis of the larynx presents a rapidly spreading, punched-out ulcer with a yellowish exudate upon a dark red base. The voice is low pitched and hoarse or raucous but rarely aphonic. Pain is not a conspicuous feature as in tuberculosis but may be present upon phonation. The tuberculous ulcer is superficial and its base is covered with a grayish exudate. It spreads rather slowly, is painful upon deglutition and the voice is weak and softly hoarse or aphonic.

In carcinoma the base of the ulcer is raised by the crowding of the deeper infiltration, it is red and constantly painful and is usually confined to the vocal cord or ventricular band on one side. The voice is continuously hoarse. It is more frequent past fifty years of age.

In lupus there is usually no pain, ulceration, edema or discharge. Dyspnea is slight or absent, the general health good and a lupoid lesion is usually present upon the skin. The epiglottis is usually involved first.

later the vocal folds and the back wall. Pale infiltrations with small granulated surfaces without ulcers are the rule. The lungs are rarely involved.

In chronic laryngitis ulceration is absent. Pain and aphonia are rarely present. The chest is usually negative and examination of the sputum does not reveal the tubercle bacilli. It usually yields to treatment.

In actinomycosis pain and dysphagia are rarely present. Sputum, as a rule, is absent and the lungs are negative. The ray fungus is found. Lesions in the nose, mouth and pharynx are usually found.

Laryngitis sicca may be confused. The presence of crusts with dry, glazed membranes are present as a rule. Ulcers are seldom present. In rare instances pachyderma laryngis, scleroma and leprosy must be ruled out.

The accompanying table as given by Frank R. Spencer<sup>1</sup> may help in making the differential diagnosis.

TABLE OF DIFFERENTIAL DIAGNOSIS (F. R. SPENCER)

Signs, symptoms and tests	Laryngeal tuberculosis	Syphilis	Carcinoma	Actinomycosis	Lupus	Chronic laryngitis
Hoarseness	Early	Early	Early	Frequent	Frequent	Early
Aphonia	Late	Late	Late	Rare	Rare	Rare
Early pain	Late	Frequent	May occur	Rare	Rare	Very rare
Late pain	Frequent	Frequent	Very often	Rare	Rare	Rare
Dysphagia	Early	Early	Early	Rare	Rare	Late
Odynophagia	Late	Late	Early	Rare	Rare	Late
Bacteria	Tubercle bacilli	Spirochetes pallida		Ray fungus	Tubercle bacilli	Mixed infection
Presence of sputum	Present	Rare	Rare	Rare	Rare	Present
Lungs	Pulmonary T. B.	Negative	Negative	Negative	Negative	
Blood Wassermann	Negative	Positive	Negative	Negative	Negative	Negative
Spinal fluid Wassermann	Negative	Positive	Negative	Negative	Negative	Negative
Röntgenogram of lungs	Positive	Negative	Negative	Negative	Negative	Negative
Biopsy	Tubercles	Gummata	Carcinoma	Ray fungus	Granuloma	Chronic inflammation

If a biopsy specimen is taken the area should be cauterized subsequently to prevent dissemination.

**Prognosis.**—The prognosis in laryngeal tuberculosis is grave, though not necessarily fatal. Under appropriate treatment the percentage of recoveries is increased. Cooper and Benson<sup>2</sup> in 798 autopsies on patients who had died of pulmonary tuberculosis found tuberculosis of the larynx in 23.3 per cent, and healed laryngeal lesions in about 4.8 per cent. These results are similar to those that have been reported at other times. As a rule, however, the patient may be expected to live only for a comparatively short time—a few months or years. Death may occur from inanition, suffocation, or hemorrhage.

<sup>1</sup> Ann. Otol., Rhinol. and Laryngol., 37, 217, 1928.

<sup>2</sup> Am. Rev. Tuberc., 25, 186, (February) 1932.

**Treatment.—General Treatment**—The general treatment of laryngeal tuberculosis is the same as for pulmonary tuberculosis. The cough may be relieved by  $\frac{1}{4}$  to  $\frac{1}{2}$  grain of codeine every two or three hours as necessary. The results obtained from the sulfonamides have not been satisfactory. Large and prolonged dosage with penicillin gives some hope of successful treatment.

The institutional treatment of tuberculosis has superseded, to a great extent, the climatic treatment. If a different climate is chosen, it should be dry, with little fog, as dust free as possible, and in a wind-proof location. Precipitation should not be too frequent. However, to send patients away from home with very limited means often results in poorer living conditions and poorer medical attention than they could have had at home, with the effect that they are worse for having made the change.

**Vocal Rest for the Larynx**—This is just as important in treating laryngeal tuberculosis as rest in bed is for the pulmonary type. It may be obtained by limiting the use of the voice to a short period each day or by instituting a strict pid and pencil regimen.

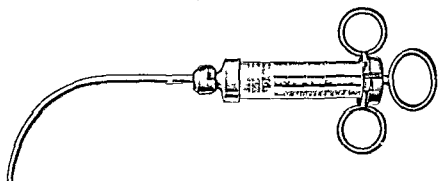


FIG. 284.—Laryngeal syringe

**Local Applications**—Innumerable remedies are recommended for the cure and relief of laryngeal tuberculosis, among them being the following:

For the relief of pain: Spraying the larynx with a 0.5 per cent solution of cocaine. If there is painful deglutition, a 2 to 8 per cent solution of cocaine may be applied locally, just before eating. Insufflations of orthoform powder may relieve the pain, is non-poisonous, and its effects last longer than those of cocaine. Spraying the secondarily infected ulcers with sulfanilamide powder relieves the pain to a great extent according to Myerson.

The local applications of formaldehyde to the larynx is recommended by many. Gallagher was one of the first to report beneficial results from this treatment. It should be used in a gradually increasing strength from a 0.5 per cent to a 10 per cent solution. The patient may be intrusted with a 1 to 500 solution for home treatment, but greater strengths should be applied by the attending physician.

Dilute aqueous solutions of lactic acid have some value in treating superficial ulcers. It may be used in gradually increasing strengths of from 5 to 30 per cent applied to the larynx by cotton mounted applicators.

*Chaulmoogra oil* gives promising results in cases in which there is dysphagia and pain in the throat. It exerts an analgesic action on the larynx which becomes more complete after repeated treatments, a pleasing sensation of warmth in the throat and chest remaining for some time following each treatment.

According to Lukens the oil works best by intratracheal and intralaryngeal injection. 1 cc. of a 25 per cent preparation of the oil in olive oil is drawn up into a Luer syringe armed with a metal eustachian catheter. While the patient holds the tip of the tongue the syringe is introduced guided by the throat mirror into the pharynx above and behind the epiglottis care being taken not to touch any portion of the mouth or throat. Two-thirds of the contents of the syringe is discharged drop by drop into the trachea while the patient breathes quietly. The remainder is then dropped on the cords while the patient phonates. Cough following injection is very slight and often absent.

**Galvanocautery**—During recent years the use of galvanocautery in the treatment of laryngeal tuberculosis is gaining in favor as a useful and positive method of treating this disease. The action of the cautery is beneficial because it tends to promote the formation of scar tissue and not because of actual destruction of the diseased area or the tubercle bacilli.

Hirsch<sup>1</sup> believes the action of diathermy or electrocoagulation has an even greater tendency to form scar tissue than cauterization because its action extends more deeply into the tissues.

Cauterization is indicated in diffuse infiltration, persistent local ulceration, areas of pseudo-edema and the base of a tuberculoma following its removal. It is contraindicated in acute or subacute cases with marked edematous swelling of the epiglottis. In late stages of the disease with extensive laryngeal involvement, particularly if the chest condition is bad, the use of the cautery is hardly indicated.

The most promising patients are those in whom the laryngeal disease is restricted to the intrinsic larynx, i. e. the vocal cords, the interarytenoid and the arytenoid regions.

Before cauterizing perfect local anesthesia is necessary. This may be done by spraying the pharynx and larynx with a 5 per cent aqueous solution of cocaine. After a few minutes powdered cocaine may be applied to the larynx on a cotton wound applicator or a 10 per cent solution may be dropped into the larynx from a laryngeal syringe.

A view of the area to be cauterized may be had by introducing a Jackson or Moshier laryngoscope, Haslinger's directoscope or Lynch's suspension apparatus may be used if desired. St. Clair Thomson prefers the indirect method of applying the galvanocautery under local anes-

<sup>1</sup> Munchen med. Wchnschr. 72: 934, 1925.

these: A fine platinum point is used at white heat at the edge of the ulcer. According to Spencer<sup>1</sup> Rather long sharp cautery points are best. These should be heated to a white heat and plunged deep into the lesion. If the point is red the surface is seared without reaching the tubercle. Fibrosis is the purpose of cauterization in order to destroy the tubercle ultimately, not total destruction at the time of the cauterization. The cautery electrode should be introduced into the lumen of the larynx and held near the tubercle. The current should be turned on and the platinum point pushed into the tubercle as soon as the point is at white heat. At a white heat it is easily used and withdrawn without adhering to the burned tissue. Two or three areas can usually be cauterized at one time or even more if the patient behaves well. If the patient's general and pulmonary condition will not permit much to be done or if the patient is not cooperative it is far better to do too little rather than too much. Normal areas of the larynx should be carefully avoided.

The joints above the arytenoids should be shunned. Subsequent fibrosis with contraction will produce ankylosis and result in fixation of one or both cords.

Isolated lesions of the epiglottis and large granulations around the interarytenoid space in many instances are successfully treated by cauterization. If removed with a laryngeal punch forceps the wound should be cauterized. Very little if any after-care is required as the reaction following the use of the electric cautery is as a rule not severe.

The cauterization can be repeated after a few weeks or months depending upon the indications.

**Heliotherapy**—More attention has been paid during recent years to the treatment of laryngeal tuberculosis by heliotherapy. The light rays have been applied generally and locally the sun rays being used and also the Linsen light. Strindberg was probably one of the first to treat laryngeal tuberculosis by the Linsen light. The entire body is exposed for the first few days for ten or fifteen minutes. The time is slowly increased up to the full time of two and a half hours every alternate day. The light bath should not be used to the exclusion of other remedies for laryngeal tuberculosis.

The Wessely machine for treating tuberculosis of the larynx is a modified carbon water cooled quartz lamp. The patient is treated with this lamp by means of the direct laryngoscope or indirectly by means of an all metal laryngeal mirror. The number of treatments Spira<sup>2</sup> found necessary ranged from 40 to 100 and the cures must be more or less selected (Wood)<sup>3</sup> Healing was obtained in 11 per cent and improvement in 34 per cent.

Local application of light into the larynx as practised by Foster and Chapman<sup>4</sup> consists in reflecting sunlight into the larynx by a mirror made

<sup>1</sup> Ann Otol Rhinol and Laryngol 37 215 1928

<sup>2</sup> Monatschr f Ohrenh 68 403 (April) 1934

<sup>3</sup> Arch Otolaryngol 23 211 (February) 1936

<sup>4</sup> Col Med 22 93 1925

up of an alloy of aluminum and magnesium. This alloy is supposed to absorb some of the heat rays while reflecting the actinic rays. The apparatus consists of a standard which fastens to the back of a chair or stand and has two mirrors—one made of the alloy for reflecting the sunlight and the other made of glass which enables the patient himself to control the direction of the sunrays. A small laryngoscope made of the aluminum and magnesium alloy is used by the patient to reflect the rays into his larynx. Samengo uses a nickel laryngoscope for the reflection of the sunrays.

The larynx is exposed to the rays for one-half to one minute each day for about a week then the exposure is gradually increased to not more than ten minutes for each day.

Plum<sup>1</sup> believes that the beneficial action of light in tuberculous lesions is a general reaction and can be obtained by general light baths.

According to Gleitsmann the Finsen light and the ultra violet rays are less penetrating than the roentgen rays and yet the latter has not produced the expected results in laryngeal diseases. The bacilli are at first increased and only after a prolonged use of a low vacuum tube is improvement noticeable. The Cooper Hewitt light or mercurial waves, the quartz lamp, the actinolight and the leukodescent lamp may be used to relieve the pain.

Ultra violet irradiation in tuberculous laryngitis may be given by means of a Kromayer lamp using a curved quartz glass applicator. Following complete anesthesia of the pharynx and larynx the epiglottis is hooked forward with the tip of the quartz applicator. Direct observation of the area irradiated is obtained by means of a small postnasal mirror. Only non progressive cases without an evening rise in temperature are suitable for this form of therapy.

**Curettage and Excision.** Curettage should be limited to the ulcerated areas while the parts which are simply infiltrated and have an unbroken surface should be carefully avoided. It has been shown conclusively that the infiltrated areas may remain quiescent indefinitely. When the tuberculous ulcer has been curetted the sluggish process stimulated and the overlying necrotic tissue removed the local treatment given in the preceding paragraphs should be continued.

Excision of a tuberculoma has a useful place in the surgical treatment of laryngeal tuberculosis when the mass is interfering with respiration. The bleeding base should be cauterized to avoid postoperative hemorrhage and infection (Spencer).

**Amputation of the Epiglottis**—Amputation of the epiglottis has yielded good results in the relief of pain and the greater ease with which patients swallow. These are cases in which there is extensive involvement of the epiglottis but with little extension into the aryepiglottic folds.

**Technic**—The epiglottis and the base of the tongue should be thoroughly anesthetized.

The amputation may be done by the direct or the indirect method. The indirect method is simpler especially if some type of a punch forceps

is used. Some laryngologists prefer to use a cold wire snare or the galvanocautery snare. If the punch forceps are used the epiglottis can frequently be removed in one piece.

Hemorrhage as a rule is not common. If present a hemostat or the galvanocautery may be applied.

**Injection of Superior Laryngeal Nerve.**—Relief from pain is obtained in a fair percentage of cases by the injection of 80 per cent solution of alcohol into the superior laryngeal nerve, either from the outside or through the pyriform sinus. This is a justifiable procedure in late cases when pain is the predominating symptom. The patient is placed on a table with the head slightly thrown back or a sand bag or pillow under the shoulders so as to stretch the front of the neck.

The superior laryngeal nerve can usually be located about 3 cm. from the thyroid notch as the nerve passes over the upper border of the thyroid cartilage before it enters the larynx. The needle is inserted at an angle to the surface of the thyroid cartilage to a depth of  $1\frac{1}{2}$  cm. The point of the needle is moved about until the nerve is struck at which time the patient will complain of a sharp pain in the ear, jaw, neck, and arm. Occasionally this pain will not be noted. The opposite side may be injected at this or a subsequent time.

The anterior portion of the epiglottis and the subglottic space are not affected by this procedure.

**Resection of the Superior Laryngeal Nerve.**—Resection of the superior laryngeal nerve was first resorted to by Avelis in 1909 for the relief of dysphagia in laryngeal tuberculosis. It is indicated if other methods have failed and the patient is in fairly good general condition. If the disease has spread to the areas not supplied by the superior laryngeal nerve, relief will be incomplete.

**Technic.**—The skin is infiltrated with 1 per cent solution of procaine hydrochloride. An incision, from 2 to 3 inches long, is made extending from the middle of the thyroid cartilage to the inner border of the sterno-cleido-mastoid muscle running parallel to the greater cornu of the hyoid bone. The omohyoid muscle is displaced toward the mid-line and the sterno-cleido-mastoid muscle is retracted backward. The lateral end of the hyoid cornu is identified and the superior thyroid artery is exposed by blunt dissection. The superior laryngeal nerve is seen lying above and anterior to the superior thyroid artery on the thyrohyoid membrane. The internal branch is picked up and sectioned. If both sides are operated on at the same time difficulty in swallowing may occur.

**Collapse Therapy.**—The various types of collapse therapy, including pneumothorax used for pulmonary tuberculosis frequently has a beneficial influence on the laryngeal involvement as the progress of the laryngeal lesion corresponds closely to that of the lung.

**Tracheotomy and Gastrostomy.**—These procedures have been suggested in rare instances where other means have failed for the purpose of putting

the larynx at rest. They might be justified in extreme cases where the patient is unable to eat or drink.

### TUBERCULOSIS OF THE NOSE

**Etiology**—Tuberculous infection of the nose is rare. When present it is usually found to be a smooth tumor formation (tuberculoma) consisting of granulation tissue with giant cells. The growth is nearly always attached to the septum. A low grade slightly depressed ulcer on the anterior portion of the septum or floor of the nose may be present or a sessile wart like tumor in which the tubercle bacilli are present.

**Pathology**—The pathologic changes found are Granulomata superficial ulceration and wart like or sessile tumors.

The granulomata are the most common.

The wart like growths are hyperplastic and like the ulcerous variety bleed easily. The removal of either variety is followed by rather slow healing and by subsequent recurrence.

The complications are perforation of the septum with extension to the skin of the upper lip and in extremely rare instances to the nasal accessory sinuses.

**Diagnosis**—The differential diagnosis must be made from syphilis and lupus. The tuberculous process tends to involve the cartilage while syphilis is more likely to affect the bony structures as well. The Wassermann reaction will assist in the diagnosis. In lupus the slow progress of the disease, the nodular appearance of the infiltration and the skin involvement will help to differentiate.

**Treatment**—The treatment consists in roentgen ray diathermy, curettage and the application of arsenical paste. The ulcer or tumor should be anesthetized with a 5 to 10 per cent solution of cocaine after which the diseased area should be thoroughly curetted or cauterized. A light application of the arsenical paste may then be made to insure the destruction of remaining fragments of tuberculous tissue. The Wessely or Finsen light or some other source of radiant energy may be tried. Diathermy may be used for the destruction of the tuberculous tissue.

In spite of all forms of treatment there is a strong tendency for the tuberculous lesion to persist and if it disappears to return.

### TUBERCULOSIS OF THE PHARYNX AND THE FAUCES

Primary tuberculosis of the pharynx and fauces (with the exception of the tonsils) is rare and is probably always secondary to pulmonary or laryngeal tuberculosis. It is usually associated with and is probably an extension from tuberculous laryngitis. It has no point of attack but may begin in the soft palate, uvula, tonsils, lingual tonsils or the pharyngeal mucosa. Unlike nasal tuberculosis it tends to spread to adjacent parts.

The part affected presents a worm-eaten appearance, the ulcers being surrounded by an area of congestion. The ulcers are superficial and



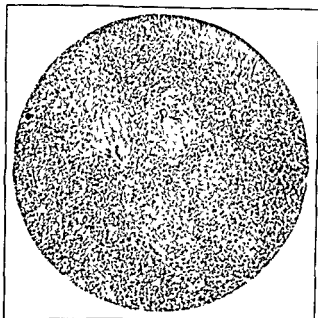


FIG. 285.—Tuberculosis of the tonsil. Extensive fibrosis with giant cells (Evanston Hospital Laboratory).

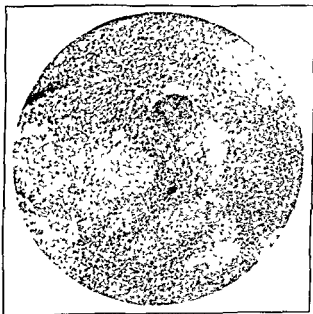


FIG. 286.—Tuberculosis of an adenoid. Note the extensive fibrosis with some necrosis and a giant cell (Evanston Hospital Laboratory)

covered with a dirty grayish secretion. They bleed easily upon probe pressure. There is little or no induration except at the borders of old chronic ulcers.

### Tuberculosis of the Tonsil

Numerous cases are reported in which the faucial tonsils are the seat of primary infection and infiltration. It is perhaps impossible to estimate the proportion of cases that are primary in the tonsils though it is perhaps larger than is generally supposed. Most writers give an incidence of from 0.5 to 4 per cent of positive cases. In other portions of the pharynx and fauces it is rarely primary.

When the pharynx is secondarily infected from the lungs it is usually from contact of the infected sputum with the mucous membrane.

When the lingual or faucial tonsils are the seat of ulceration the depth of the ulcer is great even the whole tonsil may be destroyed.

**Symptoms**—The symptoms vary with the anatomic location and extent of the lesion. If the soft palate is involved the proper approximation of the palatal muscles to the posterior wall of the pharynx is interfered with and fluids and solid food may enter the nose upon deglutition. The same condition allows the secretion to accumulate and dry in this portion of the pharynx which leads to choking and nausea in the effort to dislodge them. An infiltration of the uvula may cause pain and tickling cough. As the secretions are thick and the parts often exceedingly painful upon movements the secretions are often allowed to accumulate. The voice is muffled and hoarse or aphonic.

**Diagnosis**—Syphilis is about the only disease with which tuberculosis of the pharynx may be confounded. The following table will aid in the diagnosis.

#### *Tuberculous ulcers*

- 1 Superficial moth-eaten surface
- 2 Milky red areola
- 3 Ragged ill-defined edges
- 4 Indistinct demarcations
- 5 Grayishropy secretion
- 6 Scanty secretion

#### *Syphilitic ulcers*

- 1 Deep red anular surface
- 2 Angular areola
- 3 Sharply-cut edges
- 4 Distinct demarcations
- 5 Purulent yellow secretion
- 6 Profuse secretion

**Prognosis**—The prognosis is grave. In those cases in which it is primary in the tonsils it is not serious. When we remember that tuberculosis of the pharynx is nearly always secondary to pulmonary involvement the gravity of the disease is apparent.

**Treatment**—The general treatment is the same as for pulmonary tuberculosis.

The local treatment as given for tuberculosis of the larynx would apply in most instances to tuberculosis of the pharynx. Curettage followed by the application of pure lactic acid is a common form of local treatment. The local application of a 2 to 10 per cent solution of formaldehyde should be tried as in laryngeal tuberculosis. The pain should be controlled by the local application of cocaine the administration of

opiates, etc. The primary tuberculous tonsils or adenoid should be removed.

### LUPUS OF THE NOSE AND THROAT

**Definition.**—Lupus vulgaris is a chronic disease of the skin and mucous membrane, characterized by the formation of nodules of granulation tissue. It passes through a number of phases, and terminates by ulceration or atrophy with scar formation. It seems to be an attenuated form of tuberculosis.

**Etiology.**—Lupus of the nose and upper air passages is practically always associated with, or precedes a lupoid condition of the skin of the face.

Lupus of the pharynx and larynx occurs in from 10 to 20 per cent of the patients with lupus of the skin.

It attacks both sexes equally. It is more frequent in the country than in the city. It is most common in middle life, though it occurs at all ages. An abraded or diseased mucous membrane predisposes to its development.

**Pathology.**—Lupus of the nose generally begins on the anterior portion of the cartilaginous septum or upon the skin around the nasal orifice. It may spread from the septum to the inner wall of the ala. Four types of lesion may be recognized: (a) Granulations, (b) vegetations or proliferations, (c) tumors; (d) ulcerations. It appears as small nodules which coalesce and ulcerate, and may disappear by absorption. The reparative process takes place but feebly at the margins of the ulcer, thus forming a pale-bluish, smooth cicatrix. The ulcers reappear and then disappear. This process may continue for years without spreading to other regions. The nodules are firm and well marked. The disease rarely attacks the cartilage and never the bones.

**Symptoms.**—While lupus is due to the tubercle bacillus, there is a clinical distinction between it and tuberculous ulceration. Lupus is slow and insidious in its development, and is not necessarily associated with pulmonary tuberculosis. It has a tendency to heal, cicatrize, and recur, and does not often result in death from pulmonary involvement. One or both nostrils may be affected, and there may or may not be stenosis. The discharge varies with the stage of ulceration. At the onset it is thin and watery, and later becomes thick and even fetid, especially after crusts appear. Pain and tenderness may be present, but are usually absent. Itching is sometimes noted.

Deformity may be present if the alæ are involved, when limited to the septum deformity is rarely present.

**Treatment.**—The roentgen ray treatment of lupus has supplanted, to a large extent, other forms of therapy. Spontaneous recovery may take place, though this is exceptional.

Local escharotics, curettage, the galvanocautery, serum therapy, and surgical removal, have all been tried with varying success. The escharotics which have been used are lactic acid, carbolic acid, chromic acid, arsenic paste, and other destructive chemical agents. Curettage fol-

lowed by the local application of an escharotic afford somewhat better results though even this is far from satisfactory. In slight cases when the disease is readily accessible the galvanocautery is used with good results. Tuberculin has been attended with some success. Surgical removal by excision of the diseased area is as ineffectual as the measures just mentioned other than the roentgen ray.

### LUPUS OF THE AURICLE

Lupus of the auricle manifests itself in all the forms found in other parts of the body namely hypertrophic macular papillary and ulcerous and is usually an extension from the face.

It begins with tubercles the size of a pin head or larger which are brownish in color and slightly scaly on their surface. They are arranged in groups and are surrounded by a slight efflorescence. The skin is contracted around the diseased areas. The scarred appearance is due to the deep penetration of the tubercles. Keloid formations are quite common.

The ulcerous type is rare and is characterized by ulcerations covered with thick crusts beneath which there is a spongy base. The edges of the ulcers are undermined and pale with an occasional typical nodule.

**Treatment**—The treatment of lupus has been so uniformly successful under the roentgen ray that the older methods of treatment have become almost obsolete.

Hollander reports excellent results following the application of hot air to the diseased surfaces. The method is worthy of trial especially if the roentgen ray and ultra violet light treatments are not available.

If simpler methods of treatment fail the lupous areas of the auricle may be excised and a subsequent plastic operation performed to overcome the deformity resulting from the primary operation.

### SYPHILIS OF THE NOSE

Syphilis is a general infection of the blood and lymph streams. It is the presence of the spirochetes in the perivascular lymph spaces that excites the syphilitic reaction. This consists of an accumulation of mononuclear cells chiefly lymphocytes and plasma cells. The new tissue is highly vascular in comparison with the avascular lesion of tuberculosis. Swelling of the endothelium lining the capillaries may cause narrowing or obstruction. In later stages necrosis is frequent (gumma formation) and is associated with the presence of giant cells which however are not as numerous as in tuberculosis. The most characteristic feature is the perivascular accumulation of lymphocytes and plasma cells in the early stages and of gummata formation in the later.

A primary lesion in the nose is rare.

When diagnosed it may be found just within the alæ at the line of mucous membrane on the line of the vomer junction with the cartilaginous septum at the edge of the vertical plate or even more obscured

Picking the nose, improperly sterilized instruments or less frequently perverted sexuality are the most frequent causes.

Due to the exposed position the appearance of the primary lesion may be modified greatly. Scarlet (quoted by Babbitt) lists the change in appearance as erosive, neoplastic and scabbed or impetiginous.

In the erosive form the curmine red ulcer is round or oval with a flattened surface. It may be up to a centimeter or more in diameter.

In the neoplastic form the surface erosion 1 to 2 cm. in diameter is convex with the center elevated 2 to 3 mm. It is uniformly red or dotted with gray points giving it a papular appearance. It has a firm feeling when touched.

The scabbed form is difficult to diagnose. The surface is irregular and partly or completely covered by scabs. It is variable in color.

Secondary syphilis of the nose may manifest itself by a syphilitic rhinitis. It is not always recognized, it being regarded as a simple obstinate cold in the head. The scanty thick discharge with stenosis of the nose should however excite suspicion of the true nature

FIG. 1. Central syphilis of the nose.

of the disease. It may occur alone or with a syphilitic pharyngitis or laryngitis.

Secondaries appear in the nose and throat in the form of erythema or mucous patches. Being superficial ulcerations they are often overlooked.

*Tertiary lesions of the nose are common occurrences.*

Gummata are seen more frequently in the nose or hard palate than in the pharynx.

Seen early, the gumma of the septum appears usually over the vomer as a smooth circumscribed swelling covered by a generally inflamed mucosa. It may appear anywhere along the upper respiratory tract from the vestibule of the nose down to the subglottic space. In the early form is a gumma or a chondritis and later as an ulcer circumscribed deep and destructive or superficial and serpiginous. The favorite seat of gumma is the bony septum although they are found also on the hard and soft palate, the posterior wall of the pharynx, the epiglottis and its folds. Usually there is no sharp outline to the infiltration but a gradual fading into normal tissue. Gummatous infiltrations involve the mucous membrane cartilage and bone. Infiltration of the perichondrium and periosteum may become extensive. Necrosis may follow with loss of the bony and cartilaginous portions of the septum and in some cases suppuration of the accessory sinuses.

In the submucous thickening of the septum suggesting possible

gumma. Prehn advises a roentgen ray of the septum which may demonstrate the loss of substance due to gumma.

Nodular eruption may appear on the skin of the nose resembling acne rosacea or lupus vulgaris.

The septal thickenings due to syphilis should be differentiated from tuberculosis, lupus, hematoma, and acute infections.

In tuberculosis and lupus the bone is not involved.

A hematoma of the septum produces a more diffuse bilateral swelling. The luetic membrane has a dusky bluish red tinge as contrasted with the angry red of streptococcus infection. Shrinking by cocaine and epinephrine does not cause this bluish tint or the swelling to disappear as in other acute conditions.



FIG. 288—Chancre of the pharynx.

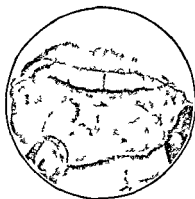


FIG. 289—Mucous patches.

## SYPHILIS OF THE PHARYNX FAUCES AND TONSILS

The fauces and pharynx are second only to the skin as sites for the manifestation of syphilis particularly in the secondary stage. This may be accounted for in part by the presence of a large number of lymphoid glands, the excessive friction, and the complex embryologic union of tissues in this region.

Congenital syphilis is more common in the pharynx than in the nose. About 50 per cent of the congenital cases show evidence in the first year of life, 33 per cent within the first six months.

**Primary**—The primary sore of syphilis occurring about the mouth is found most frequently on the lips, tonsils and tongue respectively. The tongue is usually involved on its tip or lateral edges and rarely on the posterior two-thirds. Primary syphilis on the lingual tonsil is very rare.

Females are more often affected than males and one or both tonsils may be the seat of the primary lesion.

The primary lesion is usually of short duration, though when it occurs on the tonsils the inflammation may be so great as to extend the period of ulceration to the second stage.

**Chancre of the Tonsil** Chancre of the tonsil is as a rule unilateral but may occur on both sides. Sore throat followed by swelling of the tonsil and enlargement of the cervical lymph nodes draining this area are early complaints. The tonsil is enlarged, very red in color and on palpation has a hard woody feel. An ulcer which varies in size, covered with a dirty greenish gray exudate in which the base of the ulcer is hard and indurated with an eroded border is observed. Syphilis of the

tonsil should be differentiated from acute tonsillitis, Vincent's angina, diphtheria, peritonsillar abscess, malignant disease of the tonsil and tuberculous ulcer. When the secondary skin lesions appear the diagnosis becomes definite.

**Secondary**—The secondary lesions consist of the usual erythema of the face and body and mucous membranes. They may appear from six to eight weeks after the initial lesion or even as late as several months.

The erythema of secondary syphilis may be confounded with acute pharyngitis. The differential points are the darker or dusky color (in syphilis) of the mucous membrane, the more marked involvement of the tonsils and soft palate, the diminished secretion, the line of demarcation between the inflamed area and the hard palate, the dusky symmetrical

FIG. 290—Syphilitic scars of the fauces and pharynx causing constriction of the isthmus between the nasopharynx and larynx.

patches on the anterior pillars, the opalescent appearance of the mucous membrane of the tonsils and the persistence of the disease as contrasted with the evanescence of acute pharyngitis.

**Tertiary**—The tertiary lesions appear from three to twenty-five years after the primary manifestation and may be ulcerative, gangrenous or gummatous and very destructive to both soft and bony tissues. The characteristic gummatous formations break down forming deep indurated ulcers. The palate may be partly destroyed. After healing takes place marked cicatrization occurs. A frequent site for the gumma or ulcers is at the base of the tongue.

**Symptoms**—The primary lesion may have no symptoms other than the soreness or discomfort associated with an ulcer. The cervical lymph nodes are enlarged. The chancre has a hard indurated feel.

The symptoms of the secondary stage may be absent other than a slight sore throat. Pain in the ear may be noticed if the arch of the fauces is affected. If the inflammation extends to the pharyngeal orifice

of the eustachian tube there is some deafness and tinnitus. The lymph nodes of the neck are usually enlarged. There may be cough or a tickling sensation in the throat. In some cases pain or a dull aching is mentioned. Dysphagia and a pseudomembranous angina accompanied by a slight elevation of temperature, may be present. There may also be erythematous patches on the skin and in the throat those in the throat often being mistaken for superficial ulcerations. Upon close examination they are found to be mere abrasions or elevations of the superficial epithelium.

In the tertiary stage the characteristic gummatous formations which break down forming deep indurated ulcers may be seen. The palate may be partly destroyed. The odor is characteristic and is known as syphilitic ozena. There is some pain especially upon swallowing but it is not as severe as the lesion seems to warrant.



FIG. 291



FIG. 292

FIG. 291—Syphilis of the larynx. Marked infiltration of the laryngeal cartilages.  
FIG. 292—Syphilis of the larynx with fixation of the laryngeal cartilages.

### SYPHILIS OF THE LARYNX

Syphilis of the larynx may be congenital or acquired.

The congenital type consists of a uniform and symmetrical infiltration or hyperplasia without ulceration. A similar condition may be present in the pharynx.

The primary, secondary, and tertiary manifestations of acquired syphilis may appear in the larynx though the primary lesion is extremely rare.

**Etiology**—Syphilis of the larynx is estimated as comprising from 1 to 15 per cent of all cases of syphilis. Its occurrence in the pharynx is given as about 10 per cent and in the nose as nearly 3 per cent of all cases.

Syphilis of the larynx occurs most frequently between the twentieth and fiftieth years of life. It is more common in males than females. In the congenital form it appears either in the first few months of life or at about the age of puberty. When it occurs soon after birth the lesions are usually secondary. If the second stage is completed *in utero* the



disease may only become manifest in the third stage after the lapse of several (usually from two to fifteen) years

**Pathology** The secondary stage of acquired syphilis of the larynx is usually associated with a similar process in the pharynx. The early hyperemia is followed by the formation of mucous patches.

Gummata form later which break down forming ulcers with a deep punched out appearance. The ulceration takes place very rapidly. Induration is not always present though there may be slight thickening at the edges of the ulcer. Edema is not a marked feature. At the bottom of the ulcer the cartilage may be necrosed and may be the seat of supuration that is perichondritis and chondritis of the laryngeal cartilages may be present. The anterior part of the larynx is most frequently involved. Hemorrhages sometimes occur and in rare instances endanger life.

In some instances syphilomata a granulomatous tumor may form.

As the syphilitic changes heal marked cicatrization and contractions may result. Condylomata may occur on the epiglottis or upon the laryngeal mucous membrane and cause considerable stenosis.

**Symptoms**—The vocal changes are unilateral paralysis (though it may be bilateral) with a raucous hoarseness or aphonia. Cough is in some subjects an early symptom. Pain is usually absent. If the syphilitic lesion is located on the posterior aspect adjacent to the mouth of the esophagus of the larynx dysphagia is usually a marked symptom.

**Prognosis**—Syphilis of the larynx usually yields to treatment though it may leave the vocal apparatus somewhat impaired as to its anatomic and physiologic integrity. Life is not usually in any great danger except in those cases in which the hemorrhage is unusually severe or in which the stenosis causes suffocation. When on account of the suffocation it becomes necessary to perform tracheotomy the patient should be warned that in all probability he will have to wear a tracheal tube the balance of his life.

**Treatment**—The general treatment should be as for syphilis elsewhere in the body. Local treatment to relieve the cough or pain may become necessary. In case perichondritis and necrosis of the laryngeal cartilages is present it may be advisable to remove the fragments of diseased cartilage. This may be done by direct laryngoscopy or by laryngofissure. The former is preferable as it may become necessary to repeat the operation a number of times.

In cases of extreme stenosis tracheotomy should be performed and a tracheal cannula introduced.

### SYPHILIS OF THE EXTERNAL EAR

Primary chancre of the external ear is so rare that very few cases have been reported in the literature.

The secondary manifestations may be papular pustular macular ulcerous or condylomatous. The entire auricle may be destroyed by extensive ulcerations, or it may be greatly deformed. The manifestations in the ear are usually secondary to a similar affection of the adjacent skin.

Condyloma of the meatus is rare it occurs in the proportion of about 1 to every 240 cases of general syphilis (Depres and Buck)

The course of condyloma in the external meatus is as follows In the beginning there is a red efflorescence of the skin other symptoms being absent A little later diffuse swelling of the walls of the meatus occurs The skin begins to be slightly broken and secretion is thrown upon the surface Finally warty growths of a grayish red color form in the cartilaginous portion of the auditory canal and more rarely in the osseous portion They may be large enough to block the canal

Pain usually develops with the appearance of the condyloma especially if the skin is ulcerated It is intensified by movements of the lower jaw as the glenoid fossa is in very close relation to the antero-inferior wall of the canal Deafness and tinnitus develop in proportion to the degree of the canal obstruction Fever is exceptional

Resolution may take place either with extensive destruction of the tissue or with little or no changes whatsoever In some cases the ulceration continues for many months Under general treatment resolution takes place quickly and little or no scar tissue forms Stricture of the meatus is rare

**Diagnosis** —The diagnosis should be based upon the history of specific disease elsewhere in the body the characteristic lymph node swelling the appearance of the local lesion and the laboratory tests

**Prognosis** —The prognosis of condyloma and the other secondary forms of syphilitic manifestation is favorable under antisyphilitic treatment

Gummatous formations of the external ear are usually simultaneous in their appearance with the same process in the middle ear They may appear later as deep ulcers with elevated margins

## LEPROSY

**Etiology** —It is the consensus of opinion that leprosy is communicable and due to a bacterium the bacillus of Hansen (*Mycobacterium lepre*) although it has never been conclusively demonstrated It is characterized by the presence of tubercular nodules in the skin and mucous membranes (tuberculous leprosy) or by changes in the nerves (anesthetic leprosy) At first these forms may be separate but ultimately they exist in combination In the characteristic tuberculous form there are disturbances of sensation

**Geography** —In Europe it is found in Norway the Swedish Finnish and Russian Coasts and the East sea It is more common in Asia India China Africa Egypt Abyssinia and Morocco occasional cases are found in America (California and Mexico) It is also found in Australia and the Sandwich Islands

**Modes of Infection** —There are three possible modes of infection viz

**Inoculation** —It has not been proved that leprosy is contracted by accidental inoculation though it is highly probable

*Heredity* For years it was thought to be transmitted by heredity though it is probably not

*Contagion* The disease is contagious. The bacilli are given off from the nasal secretions, open sores and the excretions of the body. Osler believes it probable that the bacilli may enter the body in many ways through the mucous membranes and through the skin. Sticker believes that the initial lesion is the ulcer upon the cartilaginous part of the nasal septum. If this is true the disease assumes greater importance to the rhinologist and suggests the advisability of maintaining thorough cleanliness of the nose on the part of those associated with leprous patients.

*Pathology* — The *Bacillus lepre* has many points of resemblance to the tubercle bacillus but can be readily differentiated from it. It is cultivated with extreme difficulty and in fact there is some doubt as to whether it is capable of growth on artificial media. *Lepra tuberosa* or tuberculous leprosy attacks chiefly the integument and the mucous membrane of the external ear, nose, palate, roof of the mouth, larynx and pharynx. The nose is a common and early site of a leprous lesion. The lesions may be in the form of a circumscribed nodule, a diffuse infiltration or a macule with pigment changes. Hyperplasia and edema may be extensive. The lesions may suggest lupus. On the skin the first changes show themselves in the form of infiltrations; the skin in one or more places over areas of several centimeters becomes elevated and assumes a brownish red or dull red color. In the region of the infiltration the sensibility disappears partly or completely and on hairy parts the hair of the affected area falls out. On mucous membranes the lesions show themselves either as small patches or tubercles or as round flat infiltrations which become ulcerated and heal with cicatricial contraction. The results are often conspicuous disturbances of the affected part: the disappearance of the cartilaginous nasal septum, the soft palate and the epiglottis. The mouth and throat present many lesions characteristic of leprosy. The tongue may contain nodules in a few cases usually at the tip and base. The alveolar areas may be involved associated with lesions of the hard palate. The soft palate and uvula may show nodules, infiltrations or ulcers. The posterior pharyngeal wall may be nodular or show ulceration or scars of previous ulcerations. Paralysis of the soft palate may occur in isolated cases. The tonsils may show nodules and infiltrations.

The epiglottis may be infiltrated or nodular or show breaking down and ulceration. Contractures may occur in advanced lesions in association with pharyngeal involvement. The epiglottis may resemble tuberculosis but has a more nodular, irregular or dull gray appearance. The aryepiglottic folds are usually involved from an extension of the lesion from the epiglottis. The arytenoids, the lateral bands and the true cords may be thickened. Ulcerations are usually not seen in the larynx other than those of the epiglottis. Stenosis of the larynx is one of the most common occurrences.

Characteristic tubercles also often develop in the bulbar conjunctiva especially at the corneal borders. The disease has a remarkably regular

and progressive course inasmuch as new lesions are always appearing. The outbreaks arise with the initial eruptions. Under febrile action the erythematous reddening of the affected parts develops and is soon followed by the formation of tubercles and nodules. At the site of the older lesions usually at the time of the fresh outbreaks changes take place and military abscesses or blebs develop either of which may end in ulceration. It is deserving of mention that at the time of these fresh outbreaks the lepra bacillus may be demonstrated in the blood in which at other times it is absent.

**Lepra Anesthetica seu Nervosa** —Anesthetic leprosy is characterized by sensibility and trophic disturbances of the skin and muscles. The formation of new tissue which produces the nodular growths of the tuberculous form is small or entirely absent. The disease begins as a leprosy polyneuritis. Anesthetic leprosy in typical cases has no resemblance to tuberculous leprosy. It usually begins with pains in the limbs and areas of hyperesthesia or of numbness. Bullae may form very early maculae appear on the trunk and extremities and after existing for a variable length of time disappear leaving areas of anesthesia though anesthesia may develop independently of the maculae. Superficial nerve trunks may be large and nodular. The bullae change to destructive ulcers. The fingers and toes are likely to contract and lose use. This type runs a very chronic course and may not be severe in its results.

Mixed tuberculated lepra is the least common form. It constitutes about one sixth of all cases about one half of which are apparently hereditary each parent often having had a different form. It begins with either a tuberculous or a non tuberculous symptom most frequently the latter are more prominent for a few months fever and the usual phenomena of tuberculization then occurring. Destruction of the cartilage of the nose sometimes ensues the soft palate also may be destroyed by ulcerations. The balance of the symptoms are a compound of the other varieties.

**Prognosis** —The disease is very chronic and progressive. The tuberculous form is destructive. The nervous form may not greatly impair the patient's usefulness.

**Treatment** —The treatment is symptomatic and general hygienic care. The local treatment of lesions of the skin consists in cauterization, carbon dioxide snow, electrocoagulation, excision and skin grafts. The intranasal application of radium may be of some value. Excision may be of value in well defined nodules of the soft parts. The nasal pack with mild silver protein causes a flow of mucus and acts as a good cleansing agent.

The recent introduction of chaulmoogra oil offers great possibilities in the treatment of leprosy and should be used in all ear, nose and throat manifestations of the disease.

Faget and Pogge<sup>1</sup> report clinical improvement in patients with lepromatous and mixed types of leprosy with promin, a sulfone preparation. The average daily dose of promin varies from 0.66 to 2 gm. The drug has also been administered by massive intravenous drip by

inhalation (in tuberculosis) and by local application. The drug causes less toxic reactions when given intravenously than when taken orally but is also somewhat less effective.

Irridectomy may be required if stenosis from contraction occurs or in rare instances from a granuloma. It is rarely required from edema.

### GLANDERS

**Synonyms**—*Lequinia malleus malleus humidus farcy morve farcin rotz*

Glanders is a contagious disease affecting horses and asses. It is communicable to man. It is caused by the *Bacillus mallei*. When it affects the mucous membrane it is called glanders and when it affects the skin and lymph nodes it is called farcy.

**Etiology**—Glanders originates in horses and asses but is communicable to man and from man to man. It is naturally more often found in men engaged in occupations which bring them in contact with beasts of burden. Though the bacillus may gain entrance through the follicles of the skin it more often does so through an abraded or a wounded surface. Cases are reported of surgeons being infected while operating upon patients who had the disease.

**Pathology**—Here are numerous closely associated nodules of low grade embryonal or granulation tissue which readily break down and suppurate. The ulcers thus formed have undermined edges which are the remnants of the wall of the preceding abscess. The process spreads by continuation though later it may be carried to distant parts. It usually appears first in the skin and then extends to the mucous membrane of the nose though it may have its origin in the mucosa. Baumgarten says it is a disease which stands midway between abscess and tuberculosis.

The nasal lesions are usually in the form of numerous closely grouped granulation nodules in the submucous tissue. There is a profuse proliferation of leukocytes and connective-tissue cells with which are admixed numerous bacilli of glanders. The proliferation continues until the pressure diminishes the nutrition of the mass especially at its center liquefaction necrosis then ensues and the nodules become abscesses. The outer wall soon breaks down and the contents are discharged into the nasal cavities. The abscesses are thus converted into open ulcers with undermined edges. Cross-sections of the masses before breaking down show them to be composed almost entirely of leukocytes connective-tissue cells and fibrous tissue. Many *Bacilli mallei* are imbedded in the masses of proliferated cells. In the acute form there are numerous polynuclear leukocytes in the adjoining tissue. In the chronic form the bone and deeper structures may be destroyed. Gangrene of the softer tissues may occur.

**Symptoms**—In the acute form the period of incubation is from three to four days. The acute symptoms often simulate rheumatism or typhoid fever in its initial stage. A little later the nodules appear either upon the skin or the nasal mucosa according to the point of infection. They rapidly increase in size until (in nasal glanders) the purulent contents

empty into the nose. The upper air passages are not often involved primarily in man. The progress of the disease is rapid and usually leads to a fatal issue in a few days or in two or three weeks.

The chronic form is fatal in about 80 per cent of the cases after six months to two years. This type bears a close resemblance to syphilis and tuberculosis. The lymph nodes of the neck are often much enlarged in the acute form. Chronic glanders often presents the symptoms of a persistent corvza. The diagnosis is difficult. It may be necessary to inoculate a white guinea pig with the nasal secretions to determine the diagnosis. At the end of two days in a positive case the testicles of the pig are swollen and the skin of the scrotum reddened. The testicles continue to increase in size and finally suppurate. After two or three weeks death occurs and the postmortem reveals nodules in the viscera. The use of mallein is highly recommended for diagnostic purposes. It is used in the same manner as the tuberculin test in tuberculosis.

In all suspected cases remove a piece of the tissue and examine sections with the microscope, make agar cultures and inject them into the peritoneal cavity of a guinea pig and watch the reactions. Also use injections of mallein and watch the results. Above all study the clinical phenomena and from all the evidence obtainable arrive at a diagnosis.

**Treatment**—In acute cases there is little hope of recovery. If seen early the tissue around the point of original infection should be either extensively cauterized or removed *en masse*. The wound thus created should be frequently bathed in a solution of zinc chloride (1 to 8). All animals and horses suspected of being infected should be killed and their bodies burned. In chronic cases tonics and potassium iodide should be given though no specific remedies are known. Roentgen ray and radium offer some hope and should be used.

*Glanders of the pharynx* is usually an extension of the same process from the nose though it may be primary in the pharynx. Nodules form here as in the nose and are attended by about the same general symptoms. The cervical and sublingual glands are early involved, break down, suppurate and discharge externally.

The chronic form is not attended with the same distinct phenomena and is often mistaken for granular pharyngitis. The nodules are mistaken for the lymphoid masses which occur in chronic follicular pharyngitis though if watched long enough they will be seen to grow gradually larger and larger until serious mechanical obstruction results. Such a process in the pharynx should arouse a suspicion of glanders and the mallein test or guinea pig experiment is given under Symptoms should be made.

*Glanders of the larynx* is rare and when present is associated with the same process higher up in the respiratory tract.

## ACTINOMYCOSIS OF THE NOSE

**Synonyms**—Lumpy jaw, holdfast or wooden tongue.

**Definition**—Actinomycosis is a parasitic infectious disease which was first observed in cattle and later in man. It is characterized by the

manifestations of chronic inflammation with or without suppuration. It often results in the formation of granulation tumors especially about the jaw and neck.

**Etiology** — Actinomycosis may be caused by any one of several species of fungi of the genus *Actinomyces* by *Actinomyces* or by *Streptothrix*. The usual exciting cause is the ray fungus of actinomyces. The predisposing causes are an abraded mucous surface or a diseased membrane. The infectious material may be carried by water or food and by straws chaff grain needles etc. The fungus probably grows upon wheat and oats hence farmers should be cautioned against chewing wheat and oat straws as they seem to be a prolific source of infection. Ligi in 1925 reported a series of 49 cases of primary actinomycosis of the tongue. Shoemakers occasionally contract the disease from the habit of holding a needle or awl in the mouth. Kissing may be the means of transmission from one person to another. It occurs chiefly in young adults.

**Pathology** — The diseased mass is made up of granulation tissue which except for the ray fungus would be mistaken for round-cell sarcoma. Epithelioid elements and giant cells are sometimes present. In the granular mass or in the pus the fungus itself appears in the form of small yellow brown or green masses about the size of a pin head which upon microscopic examination are found to be composed of a central interwoven mass of threads from which radiate club-shaped ended rays. In man the clubbed bodies are frequently absent (Senn). The histologic lesions are like in the actinomycotic nodule and in the tuberculous follicle only the germ bodies differs. Water or a weak solution of sodium chloride causes the rays to swell enormously and lose their shape, ether and chloroform have no action upon them. The gross pathologic anatomy of the disease is everywhere associated with chronic indurations with softening and liquefaction and with resulting sinuses and cysts. The head neck and especially the jaw and the cervical fascia are the sites of the disease. In the cervical fascia the disease gives the neck a brown hardness. The lymph nodes are not as a rule extensively involved. In the ox the tongue is often affected.

The lesion may be self limited as in tuberculosis by cicatricial envelopment.

The kernel like nodules are usually multiple. They may coalesce and the resulting masses may be let out. When bone tissue is affected the destruction is central while peripherally there is hyperplasia.

### ACTINOMYCOSIS OF THE PHARYNX AND TONSILS

**Symptoms** — The symptoms vary according to the part involved. The affection is chronic but occasionally runs a rapid course. The granulation tissue is abundant and the mass resembles a tumor. Previous to suppuration it is quite firm and if progressing rapidly it is surrounded by diffuse edema. Pain and tenderness are rarely present. When suppuration occurs the mass increases rapidly in size.

The frequency of occurrence in different parts of the body in 500 cases, as collected by Poucet and Berard, is as follows. Head and lungs, 55 per cent, thorax and lungs, 20 per cent; abdomen, 20 per cent, other parts, 5 per cent. In France the face and neck were affected in 85 per cent of the 66 cases reported.

The symptoms may be grouped in two classes: (a) Those referable to local tumefaction and purulent discharge, and (b) those referable to the general intoxication of the system by the suppuration products, or their metastatic spread.

The local symptoms are of slow development, and are largely those of gradual mechanical interference of the pharyngeal function. At the site, or sites, of inoculation a small rounded and reddish elevation appears, and is accompanied by the usual subjective annoyances of an attending pharyngitis. The adjacent tissue becomes swollen and tumefied, and the evidences of an acute inflammation soon change to the more permanent engorgement and solidity of a chronic condition. The swelling is irregular, but well outlined, firm to probe palpation, and not oversensitive, but slowly increases in size. Suppuration and the formation of angry-looking sinuses follow, from which issue a purulent discharge, in which are the small yellowish pellets or masses, composed largely of the typical ray fungus. The discharge is persistent, and the sinuses extend deeply and produce extensive destruction of tissue. The spread of the process does not, as a rule, occur, and it shows a tendency, if it occurs elsewhere, to do so as an isolated swelling rather than as a connected overgrowth from the original pharyngeal focus. Pain is a variable quantity, and depends largely upon the seat and extent of the peculiar swelling. Usually there is more or less continuous, heavy ache which is felt locally, and this may, at times, be eased or intensified into acute distress. Tetter of the breath and gastric disturbances from the purulent discharge are often present. The appearance of the disease elsewhere by metastasis is to be expected, especially in the lungs or the alimentary tract, though no portion of the body is free from possible invasion.

The systemic symptoms may be severe or slight, according to the degree of involvement and the exit of the suppurative products, and do not differ in their character from those usually observed in any other suppurative condition. Death occurs from slow exhaustion or through some intercurrent affection or complication.

**Diagnosis.**—Actinomycosis should be differentiated from

Sarcoma; tuberculous infection, carcinoma (of the tongue); syphilis, epulis (in jaw); and lupus.

It is, perhaps, impossible to make a positive clinical diagnosis of actinomycosis. A microscopic examination showing the ray fungus, or inoculation of a guinea-pig, may be necessary to establish it. The presence of the yellowish particles in the purulent discharge is quite characteristic, though not conclusive. Actinomycosis is probably not as rare as is generally supposed, as it is sometimes mistakenly diagnosed as sarcoma, carcinoma, osteomyelitis, syphilis, etc.



*Sarcoma* is histologically quite similar to actinomycosis. A careful microscopic examination will however in actinomycosis show the presence of the ray fungus and some giant cells. Sarcoma does not break down and suppurate so early. Both occur quite frequently in the young.

*Tuberculous disease* is attended by an enlargement of the regional lymphatics. In actinomycosis the regional nodes are not enlarged. An examination of the sputum or the inoculation of a guinea pig will show the tubercle bacilli if present.

*Carcinoma* of the tongue is usually found near the base whereas actinomycosis affects the tip. Biopsy may be necessary.

*Syphilis* in the gummatous stage is more amenable to treatment by means of the iodids. The general history of the case is also an aid in the differential diagnosis. Acute progressive actinomycosis may very strikingly resemble acute phlegmonous inflammation and osteomyelitis.

**Treatment** — The iodids are efficacious in recent cases. In old cases in which there is a mixed infection they are less efficient. The remedy should be given until marked iodism results. The injection of a 5 per cent solution of potassium permanganate into the cysts when present has produced good results. Cauterization of the skin and soft parts with the solid stick of silver nitrate is a valuable aid in those cases in which there is a fistula and suppuration. Gautier reports excellent results from the injection of a 10 per cent solution of potassium iodide into the mass. Electric needles may be inserted in the tumor and 50 milli amperes of current passed through it. Every minute a few drops of potassium iodide solution should be injected until a total of 20 minims is used. The electric current decomposes the iodide solution into nascent iodine and potassium. The chemicals thus liberated in the actinomycotic tissue act as a deterrent to the further progress of the disease. A general anesthetic should be administered for this treatment. It should be repeated in eight days.

Penicillin and the sulfonamides are effective in many cases and should be given in full doses.

The surgical treatment of actinomycosis varies from simple incision to the complete removal of the entire mass. The disease is best suited to surgical treatment before the stage of suppuration and extension to the regional lymph nodes. When it has progressed thus far it is no longer simple actinomycosis as it is complicated by a mixed or streptococcic and staphylococcic infection. A simple incision is sometimes effectual as is indeed spontaneous rupture. Should excision be resorted to it should be complete and followed by the thermocautery to prevent the spread of infection to the exposed lymph spaces. After suppuration is established treat as for tuberculosis *i. e.* curette and pack with iodoform gauze.

The disease seems to be self limited by the formation of a capsule of connective tissue and by spontaneous rupture.

### ACTINOMYCOSIS OF THE MIDDLE EAR

Actinomycosis of the middle ear is very rare and the literature on the subject is limited

In a case of actinomycosis of the middle ear reported by Majocchi of Italy the primary infection was in the lung and the infection of the ear probably occurred during a fit of coughing

### BLASTOMYCOSIS OF THE LARYNX

**Etiology**—Blastomycosis of the larynx is rare in this country but is common in South America. It usually occurs in grain workers.

A number of species of fungi may cause blastomycosis. The organism usually round or oval from 4 to 8 microns in diameter is pathogenic for both man and animal. These are ordinarily found in the sputum but may be recovered from the skin after a tracheotomy or from a biopsy specimen.

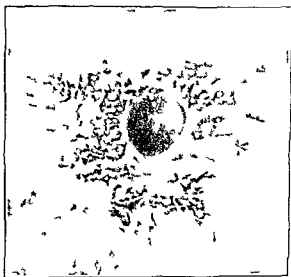


FIG. 93.—Photograph of the front of the neck of a patient showing a blastomycotic dermatitis the result of extension of the fungus from a granuloma in the larynx. Tracheotomy had been done because of laryngeal stenosis due to primary blastomycosis of the larynx. The butterfly shaped lesion was an intense dark purplish red and had a moist surface (Jackson Arch Otolaryngol).

**Pathology**—The larynx and lungs are usually involved and less commonly the pharynx, tonsils, trachea and tongue. Its appearance in the larynx varies with the extent of the disease. The early stage may be similar to any chronic inflammation followed later by infiltration, nodules and ulceration.

The cutaneous lesions resulting from infection with blastomyces are usually ragged superficial ulcers with soft granulating floors and a purulent discharge with frequent formation of thick crusts. Surrounding each ulcer is a dark red, sometimes a purplish zone.

Blastomycosis of the tongue has been reported by New and others.

**Symptoms** — The symptoms characteristic of the disease are hoarseness and a paroxysmal cough. In the beginning a burning and pricking sensation in the larynx may be noted. Dyspnea and dysphagia are later symptoms. At this stage a general debility and loss of weight and strength are present as well. The course of the disease is slow as a rule. The prognosis is bad unless adequate treatment is instituted.

**Diagnosis** — The diagnosis is made by finding the causative organism, the blastomycete, a form of yeast fungus. The differential diagnosis should be made from tuberculosis, syphilis and cancer.



FIG. 11. Photomicrograph showing at A, under high power, the blastomycete organism with a giant cell. Nuclei are numerous around the giant cell except below it B where they are absent. (B. C. Crowell) (Jackson Arch. Otolaryngol.)

**Treatment** — The treatment of choice is potassium iodide in large doses with roentgen irradiation.

### BRUCELLOSIS

**Synonyms** — Malta fever, Undulant fever.

Brucellosis is an infection due to *Brucella melitensis* and *Brucella abortus*. It is transmitted by milk contaminated with *Brucella* or by contact with infected animals.

It is of significance to the otolaryngologist because of the occasional impaired hearing or involvement of the pharynx or larynx. Impairment of hearing has been reported in as high as 20 per cent of all cases of brucellosis (Cody, Jr.).

The lesions in the mucosa of the pharynx and larynx are somewhat similar to those of tuberculous, syphilitic and certain pyogenic infections. At times it simulates acute septic infections.

The diagnosis of brucellosis is made from cultural, serologic and allergic tests. The finding of *Brucella* in cultures of the blood or excreta proves the diagnosis.

## CHAPTER XXXIII

### BENIGN NEOPLASMS OF THE NOSE, THROAT AND LARYNX

BENIGN growths of the nose, throat and larynx may be polypi or myxomas, papillomas, lymphomas, adenomas, osteomas, lipomas, xanthomas, fibromas, chondromas, angiomas and varices, amyloid tumors, cysts, odontomas, myelomas, plasmocytomas, meningiomas, lymphoid growths, teratomas, neurofibromas, schwannomas, myoblastomas, chordomas, rhabdomyomas, lingual, thyroid and mixed tumors. The latter is classed with benign growths, however, an adenocarcinoma of the mixed tumor type seems to be a low grade malignant tumor.

**Etiology** — Much has been written, while but little is known, concerning the exciting causes of these growths. Chronic inflammations seem to play an important role in their etiology. They occur at all ages, but most frequently in middle adult life. Papilloma, however, occurs more frequently in children. Both men and women are affected, but the tumors are found more frequently in men. Congenital tumors are rare.

Benign neoplasms are relatively common among street vendors, singers and speakers. The anterior commissure is the most frequent site for laryngeal tumors. Lipoma rarely occurs within the cavity of the larynx, but is located extrinsically on the anterior surface of the epiglottis. Syphilis and tuberculosis, though they produce growths of their own kind, have little influence in causing innocent neoplasms. Papilloma, fibroma and singer's nodules are more frequent than lipoma, myxoma and cysts. Heredity may be a factor of importance in the congenital tumors.

**Symptoms** — The benign tumors frequently exist without symptoms. They may be discovered only during an examination, particularly if the growth is small. Symptoms complained of most frequently are hoarseness or huskiness of the voice. At times there may be an irritation or soreness or a feeling of a lump in the throat which may cause a cough or dysphagia. If pedunculated and large they may cause an obstruction of the larynx. Angiomas may be painful because of their sensitive surface. Pain is uncommon with other benign lesions.

### MYXOMA OR NASAL POLYPUS (HYPERPLASTIC RHINITIS)

Myxoma or a nasal polyp is usually a pedunculated tumor of hyperplastic tissue which most often grows from the middle turbinate, the uncinate process of the ethmoid bone or the ethmoid cells, though it is not infrequently present in the maxillary, frontal and sphenoid sinuses. It is usually significant of a preexisting allergic or suppurative inflammation of the sinuses. In most instances it is a form of hyperplastic rhinitis of allergic origin.

**Etiology**—According to Oskar Hirsch primary polypi are created by a catarrhal rhinitis. These are rare and do not show an inclination to recur. The catarrhal inflammation of the accessory cavities creates the secondary or recurring polypi. This is the kind more frequently encountered. Many investigators believe the most frequent place for the origin of recurring polypi is the maxillary antrum and not the ethmoid as has been assumed heretofore.

One of the most frequent causes of nasal polypi is a preexisting inflammation of the membrane of the nasal sinuses and of the nasal mucosa in the region of the cell openings. This inflammation may be due to an allergic rhinitis and probably is in the great majority of instances or in some cases a suppurative infection seems to be the etiologic factor. The irritation and pressure give rise to a passive congestion and a pro-



FIG. 29a

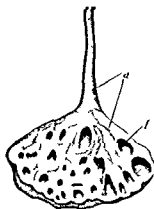


FIG. 29c

FIG. 29a—The apparently pediculated opening is inferior portion. The obstruction in the upper portion interfering with drainage and ventilation of the sinuses hence it may give rise to sinusitis or hyperplastic ethmoiditis and later to polyp.

FIG. 29c—A polyp of the cyst adenoma type removed from the nose. 4 cm long 2.5 cm wide 1.5 cm thick weight 5 grams color pink to white solid and elastic. The section shows numerous cavities filled with cholel and caseous material. Some of the cysts are lined with ciliated epithelium others have a degenerated columnar cubical or flattened epithelium and in some the epithelium is entirely lost. Some areas are infiltrated with inflammatory round cells and blood vessels. b cyst (Robert Levy specimen).

liferation of cells. A serous or edematous infiltration is a later manifestation. The connective tissue cells subsequently become filled with the serum thus leading to a hydropic degenerative change in some cells and a myxomatous or gelatinous change in others.

The tissue thus degenerated becomes pendulous and in most instances pedunculated. Such a tumor is known as a polyp.

Hyperplasia of the nasal mucous membrane due to other causes may develop into nasal polypi. If for instance a foreign body is lodged

in the nasal chamber for a long time or any other continued source of irritation is present it may result in nasal polyp.

Men are more commonly affected than women. It usually occurs during adult life but may be found in children especially the posterior choanal form.

**Pathology**—Polyps may be edematous, fibrous, cystic or a combination of these types. While polyps are usually called myxomata they are as a rule fibromyxomata. Pure myxoma is rare and when found consists of an epithelium-covered connective tissue sac which contains a mucoid fluid, some bipolar spindle cells and a fine network of connective tissue. The fibromyxoma, the usual type, is much richer in connective tissue and less so in mucoid fluid. Microscopic examination reveals all the mucous membrane elements from which they spring. The tumors are supplied with blood vessels and nerve filaments which do not penetrate the substance of the tumor but are limited to the mucous membrane covering it. They contain plasma cells which stain with polychrome methylene blue and eosin.

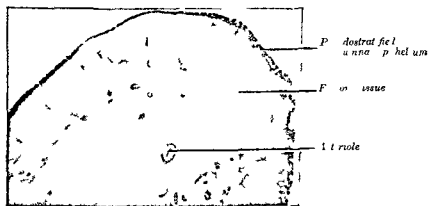


FIG. 99.—Fibrous nasal polyp.  $\times 30$

A nasal polyp is a localized edema and prolapse of the normal mucous membrane of the nose caused by allergy, an infection or by trauma. Based in some instances at least on an underlying osteomyelitis or periostitis of one of the sinuses. In those cases due to an allergic rhinitis an excess of eosinophiles can be demonstrated in the secretions or in the mucosa.

**Symptoms**—The symptoms of nasal polyps are often complex on account of the nasal obstruction and the associated inflammation of the nose and sinuses which usually coexist. The symptoms caused by the polyps are largely dependent upon their location, size and the amount of obstruction produced. If pedunculated and hanging into the lower portion of the nose they give rise to the sensation of a movable foreign body. The patient can sniff and blow them back and forth in the nose at will. If sessile they cannot be thus moved but cause a feeling of tightness or fulness across the bridge of the nose.

Nasal obstruction partial or complete is present after the polypi have reached sufficient size. The voice has the nasal twang in proportion to the obstruction produced. The voice is often muffled owing to the almost total loss of nasal resonance.

A serous or mucopurulent discharge is nearly always present in varying quantities. If the discharge is purulent or mucopurulent a coexisting sinusitis should be suspected.

Various reflex symptoms as cough and asthma may be caused by or associated with polypi. External signs of nasal polypi are not always present excepting the inclination to keep the lips parted to supplement the nasal breathing. In rare cases the tumors are of such aggregate magnitude as to broaden the bridge of the nose.

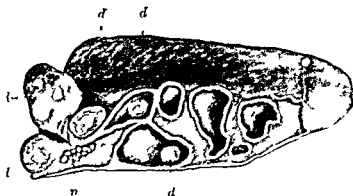


FIG. 995.—Lateral view of a middle turbinate illustrating polypoid changes. *p* polyp; *a* beginning polypoid degeneration.

The sense of smell may be impaired or lost depending upon the degree of closure of the olfactory fissure. The pharynx may be dry on account of the loss of the nasal respiratory functions or from the tenacious mucus or mucopus which is discharged into it.

Upon examination a grayish semitranslucent tumor is seen hanging in the middle meatus of the nose. If pedunculated it may move with the inspiratory and expiratory currents of air. Pressure with a probe shows a soft and yielding mass freely movable in the nasal chamber. There may be single or multiple tumors but the latter are more frequent.

**Prognosis**—The prognosis of nasal polypi is good if they are removed and the preexisting allergic rhinitis or disease of the nose and sinuses which caused them is also remedied. In those cases in which the cause is a slight nasal inflammation or one or two large polypi are present with an apparently healthy ethmoid the removal of the polypi will effect a cure.

**Treatment**—In view of the tendency of the polypi to recur the treatment should consist not only in the removal of the polypi but in attention to the various etiologic factors.

If polypi spring from the free border of the middle turbinate their removal is comparatively simple. In this location it is not difficult to engage the snare around the growths in such a way as to include also a portion of the middle turbinate from which they spring.

If they have their origin above the middle turbinate there is a strong probability that they come from the posterior ethmoid cells. It may become necessary to exenterate the ethmoid cells in certain cases.

When they have their origin in and around the hiatus semilunaris, either the maxillary, anterior ethmoid, or the frontal sinus may be the seat of infection, and it may be necessary to give attention to the affected sinus to effect a cure.

**Technic of Removal**—Local anesthesia is sufficient. This is obtained by mounting a thin pledget of cotton on a slender applicator dipping the cotton tip in a 1 to 1000 epinephrine solution, squeezing the excess

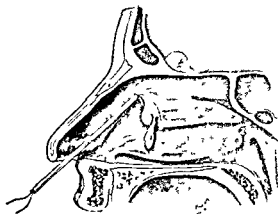


FIG. 299.—Removing a polyp and anterior end of the middle turbinate with a snare.

fluid from the cotton tip and then touching the moistened tip to cocaine flakes. The flakes are then applied to the operative field until anesthesia is produced. This may require two or three applications. Instead of using cocaine flakes, a 5 per cent solution of cocaine may be substituted. In this latter method the solution may be applied on a thin pledget of cotton adjusted over the operative field. The pledget should be left in position for from three to five minutes.

Carefully inspect the polypi by the aid of reflected light, and determine as nearly as possible their points of attachment.

With a large blunt probe the points of attachment and the neighboring parts should be examined for bare, rough bone.

The wire loop of the snare should now be introduced, so as to encircle the pendant tumor. It should be held so that both sides of it are against the septum, the lower portion of the loop being on a level with or lower than the inferior portion of the polyp. It should then be turned so that its inferior part passes outward under the polyp, and then in an upward



direction until the polyp is encircled. The procedure is often facilitated if the loop is also moved lightly in a forward and backward direction while engaging the polyp. When the polyp is engaged the snare loop is closed severing the pedicle.

If the growth is on the anterior portion of the turbinate it is usually easy, if thought necessary, to include a small portion of the turbinate (Fig. 219).

The remaining polypi are removed in the manner just described by repeated introductions of the snare.

Very little after care is necessary. If the nose becomes blocked from swelling of the turbinates an aqueous solution of 1 per cent ephedrine may be prescribed for temporary use.

### POLYPS OF THE LARYNX

The small usually pedunculated non neoplastic vascular polyp originates from the subepithelial (Reinke's) space around the free margin of the anterior third of the vocal cord or at times in the anterior commissure.

It is more common in males usually between twenty-one and sixty years of age. The size varies from a pin point to 6 mm. or more in length.

The epithelium covering Reinke's area is much thinner than over other portions of the cord. This subepithelial space may become irritated or edematous from vocal abuse or strain with the development of a simple laryngeal polyp.

The symptoms are an intermittent or constant hoarseness usually of slight degree. Vocal fatigue or a sensation of a foreign body may be noticed. Dyspnea is rare unless the polyp is of large size.

The polyp should be differentiated from a vocal nodule, papilloma and granulomatous or malignant growths.

The treatment is directed to any etiologic factor discovered and to the removal of the polyp by the indirect or direct method.

### POLYPS OF THE SEPTUM

Polypi (see Angioma) originating from the septum are angioma, fibroma or granuloma. Pissow and Glos both consider polypi of the septum to be a tumor like formation which develops on an inflammatory base.

Schreyer examined 13 polypi of the septum in the clinic in Breslau and came to the conclusion that they are abnormally proliferating angiomas. As a therapeutic measure Schreyer recommends excision of the adjacent mucous membrane of the septum and cauterization of the spot of attachment to prevent recurrences. He does not believe that these formations are of an inflammatory nature and considers them abnormally proliferating angiomas which become ulcerated on the surface and secondarily inflamed.

## PAPILLOMA

**Papilloma of the Larynx.**—**Etiology.**—Multiple papillomas (papillomata) of the larynx and adjacent parts usually occur in early childhood with a marked tendency to recur and to be transplanted to neighboring parts. Papillomas, occurring in adults, do not have this tendency to recur to a great degree.

The disease is not common. Large clinics report seeing only 1 or 2 cases a year. It is reported to be more frequent in boys than in girls. The etiology is not known, but a virus is thought to be a factor.

**Pathology.**—Papillomas are formed from thickened, projecting finger-like masses of stratified squamous epithelium containing cores of connective tissue, which bears the blood-vessels. The epithelial basement membrane is well demarcated without an underlying epithelial cell infiltration. The epithelial cells do not vary to a great extent in size, shape or staining properties. A rare mitosis is seen.



FIG. 300.—Papilloma of the anterior commissure of the larynx in an adult

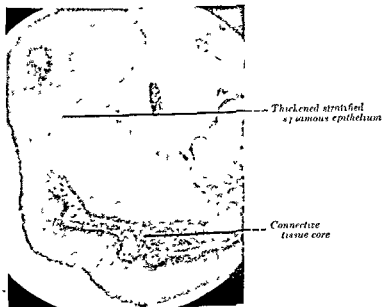


FIG. 301.—Papilloma of the vocal cord showing the thickened squamous epithelium with the connective tissue core carrying the blood-vessels, nerves and lymphatics ( $\times 80$ )

Papillomas are usually attached to the anterior third of the vocal cords or at the anterior commissure though they may spring from any portion of the larynx. Tuberculous papillary growths often grow at the posterior commissure.

Papillomas may appear upon inspection to be either pedunculated or sessile though upon microscopic examination all varieties have the same structure. It is probable that those having a sessile or diffused base are in reality only numerous pedunculated growths closely crowded together and fused in the process of development. When single the growths may present a distinct pedicle with a warty growth at its extremity. When multiple they may appear to be sessile or they may have the appearance of a cauliflower like growth.



FIG. 30. — Multiple papillomas in the larynx.

Papillomas may be pale or congested when congested they are more active in their growth.

**Symptoms**—Hoarseness or aphonia are characteristic symptoms. The

hoarseness and aphonia may be transitory or constant. Dyspnea and cyanosis are sometimes severe and when present necessitate immediate tracheotomy. If the dyspnea is great the supraclavicular region will be depressed.

The general health is often impaired and the weight diminished by several pounds.

**Prognosis**—Juvenile papillomatosis is characterized as a self limited disease as the tendency to recurrence lessens or disappears after the establishment of puberty when many cases spontaneously recover however many cases do not exhibit this tendency to a marked degree. The factor which produces this tendency to spontaneous recovery is not known.

The condition may progress to asphyxia from the extent of the growth or from fibrous stenosis.

**Treatment**—The various forms of treatment consist of the local application of estrogenic hormones, radium, roentgen ray, fulguration and diathermy, laryngo-fissure, endolaryngeal operation and tracheotomy.

**Estrogenic Hormone**—The local application of an estrogenic hormone given once or twice a week has been reported (Broyles) to have a beneficial action on the juvenile type of multiple papillomas by changing them to the adult type which is more amenable to treatment.

**Fulguration**—Fulguration and diathermy have been used extensively. The reports indicate considerable disappointment with some good reports. An acute edema may result in permanent stenosis of the larynx.

*Radium*—Many favorable reports are found in recent literature from such able men as New Harris, Freudenthal, Gilbert and others concerning the excellent results obtained from treating multiple papillomas of the larynx in children. Chevalier Jackson<sup>1</sup> is opposed to the use of radium in these growths, finding in his experience either no effect on the growth or an increased tendency to activity. Clerf<sup>2</sup> upholds Jackson in his belief, stating its use should be condemned.

The treatment carried out by New consists of radium applications both inside the larynx and outside the neck. The patients are placed under ether anesthesia and suspended by means of a Lynch suspension apparatus. No attempt is made to remove the papillomas. A small tube containing the emanations or the radium salts is inserted into the glottis and held there by means of forceps. The tube is kept moving under direct observation so that no particular area is overtreated. From 75 mg. to 150 mg. or millicuries of radium are used for from twenty to thirty five minutes or longer depending on the particular condition. No screening is used except the silver tube which contains the salt or emanation. As a rule the patients are treated about once in six weeks or two months. If recurrence is noted further treatment are given before the recurrence becomes marked.

Besides these suspension treatments radium is applied outside the larynx as a rule about 3000 mg. hours are given using 2.5 cm. of wood and 2 mm. of lead screening.

*Röntgen Rays*—Roentgen therapy for juvenile papillomas of the larynx has advanced in recent years to such an extent that it seems to give equally good if not better results than radium.

*Laryngo-fissure*—The trend of opinion is away from laryngo fissure (thyrotomy) and the indirect laryngeal method and toward tracheotomy and the direct laryngeal method if symptoms of impaired respiration are present.

Laryngo-fissure is not favored on account of the frequent recurrence of the growths. The operation is somewhat disfiguring and often attended with stenosis of the larynx and an impairment of the voice especially if a high tracheotomy is done. The chief argument against this operation for laryngeal papilloma is that other methods afford a better means of relief.

*Direct Laryngoscopy* (Chapter I VIII) with Jackson's self illuminated tube spatula, Lynch's suspension apparatus or Haslinger's directoscope is much superior to indirect laryngoscopy. The growths are brought into clearer vision and greater accessibility. Removal by direct laryngoscopy may be attempted when dyspnea and cyanosis are not present. If these symptoms are present the instruments for tracheotomy should be in readiness if suffocation occurs. Jackson advises against an attempt at removal of the base. The procedure is repeated as the growths reappear. Distortion, atresia and permanent impairment of the voice may follow too many attempts of a thorough removal.

<sup>1</sup> Bronchoscopy and Esophagoscopy p. 704 1927

<sup>2</sup> Arch. Otolaryngol. 6:345 1927

*Tricheotomy*—A low tricheotomy should be performed in all cases in which dyspnea and cyanosis are present. The procedure should not be postponed until it becomes an imperative measure but should be done while the patient is still in a condition to permit the operator to do it with deliberation and good technic. It should rarely be followed by the immediate removal of the growths. Weeks or months should usually intervene. Indeed it is useless to remove the growths while they are in the active stage as they will recur often in greater abundance than before their removal. If



Fig. 303.—Larynx showing from the front the site of tricheotomy.

the healthy tissue is injured during the operation the growth will often appear at this point.

When the growths show a state of quiescence or of retrogression they may be removed by indirect or direct laryngoscopy or through the tracheal wound.

**Papilloma of the Pharynx**—Papilloma rarely occurs on the walls of the pharynx but is common in the faucial region. It is most frequently found upon the uvula, the free borders of the pillars of the fauces and the tonsils.

They may be single or multiple, sessile or pedunculated. Behind the fauces or in the pharynx proper they are rarely pedunculated and are chiefly limited to ragged excrescences. The elevations vary from tumors as small as a pin head to those of considerable size. They often contain pearls or nests which may be mistaken for the nests or pearls of epithelioma. The cells in papilloma are uniform whereas in epithelioma they are multiform.

The presence of a papillomatous growth in the fauces or pharynx often excites a reflex cough with a sense of fulness and tickling in the throat.

**Treatment**—The tumor should be removed to its base with a knife, snare, cutting forceps or cautery. The base of the growth should be removed or cauterized with the galvanocautery. If this is not done they are likely to recur.

**Papilloma of the Tonsils**—Papilloma of the tonsil is more often multiple than single and presents the general outlines of a bunch of grapes. In some instances there may be one pedicle with many papillomas attached, whereas in others there may be many pedicles. If single and large it may be mistaken for a supernumerary tonsil. Like all papillomas it has a tendency to return.

Small papillomas in the tonsillar crypts are fairly common.

The growths as a rule give rise to no marked symptoms. A slight hacking cough, a tickling sensation and the feeling of a foreign body in the faucial region are frequently mentioned. The only change noted in the surrounding tonsillar tissue is an increased hyperemia around

the attachment of the tumor. Pain is never present. The tumors vary in size from that of a pea to a large walnut.

**Papilloma of the Nose — Etiology**—Papilloma of the nose is very rare. Kramer and Som<sup>1</sup> found reports of only eighty-six instances of true papilloma of the nose and accessory sinuses.

They are commonly found in males between forty and fifty years of age. A history of a chronic infection of the nose and accessory sinuses is obtained in many instances. Embryonal rests of epithelium in the sinuses have been suggested as the starting point for these tumors.

**Pathology**—They may arise as solitary or multiple tumors varying in size, form and consistency. They occur most frequently in the vestibule of the nose, especially on the anterior portion of the septum. They occur at times in the posterior or superior portions of the nasal cavities and very rarely as primary growths in the nasal sinuses.

Most investigators consider them as being on the border between benign and malignant growths.

Papillomas from sinuses usually have pedunculated or cauliflower forms. They grow rapidly and tend to recur quickly after removal.

The excessive proliferation of the epithelium with the somewhat less growth of the connective tissue is the major pathologic finding.

Two types of these growths have been described: (1) a cauliflower-like mass of nodules attached to the tumor base by a stalk of varying size. They have a hard feel and are somewhat movable. (2) A diffuse type in which small single nodules are found over a large portion of the nasal mucosa.

Histologically the tumor consists of an epithelial proliferation with a connective tissue groundwork.

**Symptoms**—Papillomas of the nose rarely produce severe symptoms. Years may elapse before complete nasal obstruction takes place. Secondary sinus infection may occur. Nasal hemorrhages occur at times. True papillomas, if large, may cause atrophy encroaching on the orbit and cheek. Both sides may be involved or they may appear externally or project posteriorly into the nasopharynx.

Examination reveals a growth of red color with a firmer consistency than polypi. The growths may appear as a series of deep folds in the mucous membrane. Others have a cauliflower appearance, those arising in the region of the vestibule may resemble closely the ordinary wart.

The differential diagnosis should be made from nasal polypi. The diagnosis is made from a microscopic examination of a portion of the tumor.

**Prognosis**—Destruction of tissue and bone have been observed. As a rule the prognosis is more favorable if the tumor is solitary and if located in the anterior half of the nose (Seydell). Recurrences are common after removal.

**Treatment**—Removal of the growths with destruction of the base with the cautery or diathermy is advisable. Roentgen ray and radium have proved to be of great value in the after treatment.

<sup>1</sup> Arch Otolaryngol 22:29 (July) 1935

Diathermy may be of value in the removal of the neoplasms as bleeding would be less. Radiotherapy alone has been advocated with favorable results reported.

If the growth is removed with a snare or nasal scissors the surrounding tissue should be anesthetized by the local application of epinephrine and cocaine flakes or a 5 to 10 per cent solution of cocaine after which the tumor is excised. After the bleeding has ceased the wounded surface should be mopped dry and cauterized with the galvanocautery. This is done to prevent a recurrence of the growth. When papilloma recurs in a patient forty or more years of age the possibility of carcinoma should be suspected.

### LYMPHOMA OR LYMPHADENOMA

This variety of benign tumor is the most frequent growth in the pharynx. This is to be expected on account of the widely disseminated lymphoid tissue and the numerous lymphoid vestiges. The matrix of the tumor is connective tissue in the meshes of which are aggregated the lymphoid cells. The cell groups are often crowded together and vary greatly in size. They like lymphoid tumors elsewhere have a strong tendency to multiply. They may be attended with or may follow mediastinal complications of a like nature (Villar). A single tumor especially when pedunculated at times offers some diagnostic difficulties. But when we take into consideration that the adjacent lymphatic nodes in the neck are enlarged and soft the tumor in the pharynx though pedunculated should be suspected to be lymphomatous.

**Lymphoid Tumors of the Tonsil** — A pure benign lymphoid tumor of the tonsil is very rare only a small number of such cases have been reported in the literature.

The histologic picture is similar to the hyperplastic lymphoid tissue of the tonsil. It is probable that these growths are only an unusual distribution or formation of the tonsil itself rather than true new growths.

**Lymphadenoma in Hodgkin's Disease** — In every case of Hodgkin's disease it is advisable to examine the tonsils as they may be the seat of a lymphadenoma such as is present in other parts of the body. In the early stage of the disease it may be impossible to assert positively that the tonsils are involved though they may appear abnormally enlarged. Lymphadenoma of the tonsil is only a local expression of a disseminated lesion of a similar nature throughout the general lymphatic system. Roentgen rays give marked relief.

**Lymphoma of the Nose** — Lymphoma of the nose is characterized by a smooth mass pinkish red in color, and less dense in consistency than fibroma. It is not common and a microscopic examination is necessary for a positive diagnosis. The treatment is the same as for polypi and fibroma except radium or roentgen ray may be used.

### HODGKIN'S DISEASE

Hodgkin's disease is a disease of the hemopoietic organs is invariably fatal and probably is neoplastic in nature but is considered by many as inflammatory.

The disease is most common in middle age and in men the average case lasting one to two years. Enlargement of the cervical lymph nodes is frequently the first sign of the disease. A progressive anemia is constant, but the white blood cells show no uniform change. Fever is common, often assuming the Pel-Ebstein type.

The microscopic appearance of a lesion (usually a lymph node) is characterized by its pleomorphism. Pale epithelioid cells are present in great numbers and very frequently very large or giant cells which



*Characteristic pleomorphism.  
Many eosinophils are seen  
under the microscope.*

FIG. 304.—Hodgkin's disease ( $\times 400$ )

may be mono- or multinucleated, are observed. These are known as Dorothy Reed or Sternberg cells. Eosinophils are characteristic and lymphocytes, plasma cells, and leukocytes may be seen. Necrosis may appear later and finally dense fibrous tissue may form.

### ADENOMA

An innocent adenoma is an epithelial tumor of glandular structure which closely resembles the gland from which it arises. A true adenoma is surrounded by a fibrous capsule formed by pressure on and subsequent condensation of the surrounding tissue.

Adenomas have been reported as growing from the septum ethmoid, pituitary gland, bronchi, and in rare instances from the ceruminous glands. The growth consists of a simple hyperplasia of the gland structure with a strong tendency to recur unless completely removed. They possess potentially malignant characteristics. The majority develop in women before forty years of age.

**Symptoms**—Adenomas bleed so readily upon examination with a probe that a sarcoma is suggested. A microscopic examination may be necessary to differentiate.



Adenomas of the nose in addition to the ease with which they bleed produce a rapidly increasing nasal stenosis.

Bronchial adenomas usually located in one of the main bronchi near the hilum vary in size from a small pea to that of a pigeon's egg. The tumor is usually smooth but may be lobulated and at times pedunculated. The pedunculated type grows as a rule within the bronchial lumen. The sessile or intramural type grows between the cartilaginous rings encapsulated between the bronchial mucosa and the outward fibrous membrane.

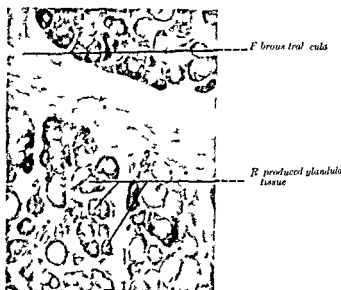


Fig. 3 — Benign adenoma of the vocal cord. The glandular tissue has no connection with the duct of the gland itself.

**Treatment** — The treatment should consist in the total removal of the tumor. In order to insure this its base should be cauterized or curetted. The bleeding which attends the removal of adenoma is considerable but may be readily controlled by nasal tampons.

Roentgen ray and radium should follow the surgical removal of this type of tumor.

The bronchogenic tumors with a pedicle may be removed successfully by bronchoscopic means. The sessile type will probably require an intrathoracic surgical intervention.

### OSTEOMA

Osteoma is a true bone tumor essentially benign in nature and frequently multiple.

**Etiology**—Osteoma of the nose and sinuses is rare. Sjoberg up to 1934 collected 343 cases. They usually occur in the frontal sinus but the other sinuses may harbor the tumor as well as the mastoid or at times the tracheobronchial tree.

About 50 per cent of these tumors develop in the young when the bones are undergoing the greatest developmental activity.

Various theories have been advanced as to the etiology of these tumors namely: (1) That they arise from embryonic cartilaginous cells at the junction of the ethmoid and frontal bones. (2) that they arise from the periosteum of the sinus walls. (3) that they originate from the diploe, (4) syphilis has been mentioned as the cause before the advent of more exact diagnostic methods. It is seldom given today as an etiologic factor. (5) inflammation within the sinus has also been advanced as a cause. The consensus of opinion at the present time is that osteomas develop from periosteal embryonal rests.

**Pathology**—Osteoma is usually composed of dense compact cancellous bony tissue on a congenital or postnatal matrix of osteoclasts and usually has its growth from the periosteum though it may grow from the medullary portion of the bone. Some osteomas are soft and spongy with a dense capsule of bone while others are dense throughout their substance. The spongy type occurs more frequently. They are in some instances pedunculated the pedicle being composed of either spongy bone or soft connective tissue and mucous membrane. They vary from the size of a small walnut to that of a goose egg.

They may occur in any of the accessory sinuses but are more common in the frontal. They may invade the nasal and orbital cavities when growing from the sinuses. The favorite points for development of osteomas of the frontal sinus is the recessus nasalis of the frontal sinus and the suture between the ethmoid and frontal bone. Osteomas of the ethmoid region originate from this latter or from the junction of the frontal and ethmoid bone. Osteomas of the antrum and sphenoid most often develop from the areas bordering on the ethmoid labyrinth. It sometimes springs from the inferior turbinate and occludes the nasal chambers.

**Symptoms**—Growth usually is slow frequently ten years or more are required before the tumor causes the patient to seek relief.

If the tumor grows rapidly the patients often have attacks of pain over the site of the growth. If the orbital cavity is involved the contents are displaced usually forward outward and downward with a marked proptosis. Diplopia and optic nerve derangement are often prominent symptoms.

In the intranasal type external nasal deformities may be present. As the nasal chambers are usually invaded nasal obstruction is a prominent symptom. The growth of the tumor externally produces more or less marked deformity and in some instances the resemblance to horns is so great that the cases are referred to as "horned men." In some instances they present the "frog face" type of countenance especially when both sides of the nose are involved in the region of the infraorbital

ridge. The tumors within the nasal cavity or sinus often project into the brain cavity.

Palpation of the tumor whether intranasal or extranasal yields a sense of bony hardness. The lacrimal duct may be occluded.

Osteoma of the frontal sinus which has extended into the brain may cause death by pressure necrosis but more frequently by secondary infection of the intracranial structures.

The symptoms of osteoma of the frontal sinus are external deformity in the later stages, headache usually an early symptom, discharge from the nose has been recorded in comparatively few instances. Shea reports a case with vertigo as the predominant symptom, convulsive attacks have been mentioned.

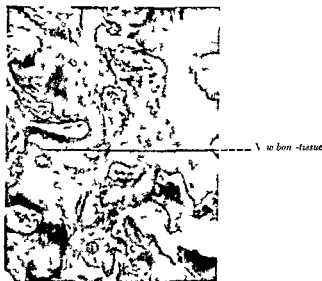


FIG. 33C. Benign osteoma ( $\times 100$ ). A benign osteoma is an overgrowth of bone. It may be cancellous or compact in structure.

Osteoma of the maxillary sinus when large enough to give symptoms would be neuralgic pain if the infraorbital nerve is involved, epiphora if the tear duct is blocked, swelling of the cheek and upper portion of the alveolus in the later stage, possibly exophthalmos (eyeball pushed upward), diplopia and impaired or loss of vision. A secondary maxillary sinusitis is common.

**Diagnosis**—The diagnosis is largely based upon the roentgen ray, the microscopic examination of the tissue and the symptoms if present. Mucocoele of the ethmoid, frontal and maxillary sinuses must be differentiated.

**Treatment**—The surgical removal of the bony growth is usually the best treatment. The technic of the operation varies with each case.

In the removal of osteoma if there is no pedicle it is better to enucleate the tumor rather than to attempt to chisel or drill into its substance.

as it is often so dense as to resist the instruments. The incomplete removal of the pedicle may result in the recurrence of the osteoma.

**Osteoma of the Mastoid.**—Osteomas of the mastoid are very rare. Simpson<sup>1</sup> in 1940 found 32 cases including 2 of his own. They may be the dense hard ebony type or a soft spongy bone with many gradations between. The majority have a bony attachment. The solitary movable type with soft tissue attachment is infrequent.

Symptoms are usually due to obstruction of the external auditory canal.

### TRACHEOPATHIA OSTEOPLASTICA

Tracheopathia osteoplastica or bone tumors of the trachea, bronchi and lungs are rare. Hiebaum<sup>2</sup> up to 1934 found 30 osteomas of the lung and more than 70 involving the trachea (Moersch, Broders and Havens<sup>3</sup>).

The multiple bony masses or nodules are usually discovered by bronchoscopic examination or at autopsy. They usually grow between the cartilaginous rings and under the mucosa. The posterior membranous portion of the trachea is free from the growths.

The symptoms, if present, are hoarseness, cough, excess secretion, at times hemoptysis (Curt<sup>4</sup>) and gradually increasing obstructive symptoms.

### OSSIFYING FIBROMA

An ossifying fibroma or secondary osteoma (Ewing) is a rare benign growth of non-dental origin involving the superior or inferior maxilla. The connective tissue of the ossifying fibroma tends to differentiate into bone. A later static phase of development may be reached.

**Etiology.**—The cause of the growth is not known. It has been assumed to be a spontaneous benign neoplasm, a reaction of membranous bone to trauma and infection, a disturbance of growth (Eden<sup>5</sup>) or a "developmental cell derangement of the dental periosteum incident to the completion of the permanent tooth cycle in the deciduous teeth" (Harris and Hagaman<sup>6</sup>). The tumor occurs, as a rule, between the ages of twenty and thirty and in women more frequently than in men.

**Pathology.**—The tumor is most frequently found on one side of the superior maxilla and may involve both sides or the same side of both jaws. The alveolar process, canine fossa or malar bone region may be invaded by direct extension, producing a characteristic facial deformity or the growth may invade the hard palate or extend superiorly to the orbit.

The histologic examination of the growth shows thin, irregular bony trabeculae scattered in a stroma of abnormal connective tissue (Hara<sup>7</sup>).

<sup>1</sup> Arch Otolaryngol, 32, 642 (October) 1940.

<sup>2</sup> Ztschr f Path., 47, 249, 1934.

<sup>3</sup> Arch Otolaryngol, 26, 291 (September) 1937.

<sup>4</sup> Ann Otol Rhinol and Laryngol, 53, 839 (December) 1944.

<sup>5</sup> Brit Jour Surg, 27, 323 (October) 1939.

<sup>6</sup> Ann Otol, Rhinol and Laryngol, 51, 505 (June) 1942.

<sup>7</sup> Arch Otolaryngol, 40, 180, (September) 1944.

**Treatment** — The treatment of choice is surgical excision preferably during the stage of quiescence followed by cauterization of the surrounding area. In selected cases small fractional doses of roentgen ray or radium may be given as a preoperative measure.

### LIPOMA

**Etiology** — Lipomas of the pharynx and larynx are rare.

Palmer and Mehler<sup>1</sup> found 32 cases of lipoma of the larynx reported during the last forty-two years.



FIG 307 — Lipoma (X 80)

Various theories of etiology have been advanced such as (1) a simple hyperplasia of fat cells (2) an invagination of mesodermic tissue which develops into a lipoma and (3) a disturbance in the lipid chemistry of the cells. Some cases have been associated with tuberculosis.

The tumors have been observed in patients of both sexes and of all ages.

**Pathology** — New and Childrey found lipomas arising from the posterior wall of the pharynx were sessile as a rule whereas those of the palate or tonsillar fossa were pedunculated. They are usually smooth or lobulated, covered by mucous membrane composed of fatty cells with or without additional fibrous tissue. In rare instances cartilaginous tissue is present.



FIG 308 — Lipoma originating from the right aryepiglottic fold

Frequent points of origin are the epiglottis, the aryepiglottic fold or the ventricle. The attachment may be by a pedicle or by a thick base.

**Symptoms.**—The usual complaint is difficulty in breathing and choking when fluids are taken. As a rule there is a gradual onset with absence of pain, fever or bleeding. A slow development of obstructive symptoms is usually noted. If pedunculated, intermittent obstruction to breathing may be noted.

Lipoma of the pharynx may reach the larynx and give rise to laryngeal symptoms.

Laryngoscopy usually reveals a smooth, lobed, pedunculated growth. The tumor appears pink, often with areas of yellow. The mass is usually soft and elastic in consistency.

Lipoma of the nose may be external or internal, and is usually pendulous. When external it generally affects the alae of the nose. The treatment consists of the excision of the growth.

**Differential Diagnosis.**—Lipoma should be differentiated from the various benign tumors and especially from vascular tumor, lymphosarcoma, cystic lesions and mixed tumors.

**Treatment.**—Removal of the tumor by direct or indirect laryngoscopy is satisfactory as a rule. An external operation may be necessary for the larger tumors. If removed incompletely recurrences have been reported.

## XANTHOMA

Xanthoma is a rare disease characterized by the deposit of lipids in the form of yellowish-brown plaques or nodules on the external and internal surfaces of the body.

A few cases involving the mouth, pharynx, larynx, trachea, and esophagus have been reported.

**Etiology.**—It is generally believed that the abnormal deposition of the lipids in the body is due to a disturbance of the fat metabolism plus some local factor in the tissues involved.

Various conditions have been associated with some cases of xanthoma such as chronic jaundice and diabetes mellitus and insipidus. However, many cases have been observed with no predisposing cause found.

**Pathology.**—The lesions may vary in size, number and location. In addition to other regions they have been observed as multiple nodules or plaques in the mouth, pharynx, larynx and trachea. They have a smooth or nodular surface with a yellow color.

Histologic and chemical examinations show that the xanthomatous nodules contain cholesterol and other fats and occasionally a number



FIG. 309.—Xanthoma of larynx. A large tumor mass as well as small nodules can be seen.

of Touton giant cells. These giant cells contain a peripheral zone of lipid-containing cytoplasm with central nuclei arranged in a circular formation. The xanthoma or foam cells are the most prominent feature.

**Symptoms**—The course of the disease is characterized by an early inflammatory stage, a later tumor formation and a still later stage of regression or fibrosis.

A general weakness is frequently mentioned. If the lesions are in the upper respiratory tract and accompanied by contractions or tumor formations interference with respiration or swallowing may be noticed.

**Treatment**—Treatment of these tumors has not been successful. Some cases have been improved by treating the associated condition when present. Other cases have shown a spontaneous recovery while others may last indefinitely. Roentgen rays have been tried. Single nodules may be removed by excision or cautery.

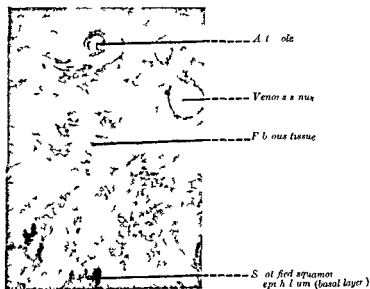


FIG. 31. Fibroma ( $\times 100$ ). Numerous arterioles, veins and venous lakes can be seen surrounded by fibrous tissue.

### FIBROMA

**Nasopharyngeal Fibroma**—**Etiology**—Nasopharyngeal fibromas usually occur between ten and twenty five years of age, more commonly in males. It is usually single although multiple growths have been reported. Softer varieties are called polypoid fibromas or a fibromatous polyp. As age advances there is a tendency for the growths to recede or undergo spontaneous cure. The true fibroma usually arises from the nasopharyngeal recess, less frequently from the anterior sphenoid wall, base of the sphenoid, nasal process of the occipital bone, nasal choanae, first cervical vertebra, mouth of the eustachian tube, posterior palatine surface and the pterygomaxillary recess. The softer fibromatous

myxoma, or "polyp," usually arises in the nasal cavity and extends into the nasopharynx.

**Pathology.**—The surface of the tumor is covered by mucous membrane under which are found large ramifying blood-vessels. The mass is made up of dense fibrous tissue and agglomerated cells, contains large venous channels most abundant in the peripheral portions. In very rare instances a myxomatous tumor may have the tendencies and aspects of a fibroma, just as primary fibromas may become mucoid in character.

Fibromata may be sessile, but are more often pedunculated. They often attain large size. Large fibromata are frequently attended with inflammatory processes, hence adhesions to the adjacent structures are common. Additional blood supply is derived through the adhesions.

They do not metastasize, but grow by extension. They may invade the orbit, accessory sinuses, pterygoid and temporal fossae or the intracranial cavity.

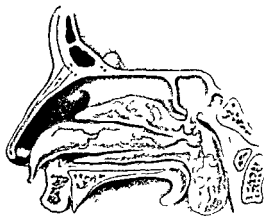


FIG. 311.—Fibroma of the nasopharynx springing from the base of the sphenoid and sending finger like prolongations into the nasal chambers. Large venous channels are spread over the surface of the tumor.

**Symptoms.**—The early symptoms are those of nasal congestion and obstruction with more or less hemorrhage. The bleeding sometimes becomes an alarming complication. The voice becomes "flat" or "dead" in quality, and respiration and deglutition are impeded as the process advances. At a later stage, there is pain and mucopurulent discharge. When the growth has attained considerable size, the "frog face" becomes well marked, the maxillary bones are separated, and exophthalmos becomes a prominent symptom. Aphrosvia and drowsiness are often present.

If the growth extends upward it may encroach upon the cranial contents and give rise to such symptoms as paralysis, etc., this is followed in nearly every instance by death.

The foregoing symptoms increase in severity as the growth extends, until the absorption of bony tissue is considerable, unless the tumor extends beyond the nasal and pharyngeal chambers, as into the cranial



cavity In this event the pressure necrosis of the bony tissue is not so great

Examination shows the tumor to be a rounded mass of a pinkish or dark purple color The veins are frequently varicose hence the examination by digital or instrumental aids should be done carefully to avoid injuring them The growth may project into the posterior nares or its direction may be toward the antrum and other sinuses Under finger pressure it is firm and elastic and if small its base may be outlined If pedunculated it is movable unless it has become fixed by inflammatory adhesions If it extends through the sphenomaxillary fissure it may be felt under the zygoma As adhesions are usually present its outline is difficult to distinguish

**Diagnosis**—The histologic resemblance to sarcoma is often so close that a differentiation is difficult unless the age sex and origin are such as to point to its fibrous nature Sarcoma is rarely or never pedunculated whereas soft fibroma is frequently so Hard fibromas are usually sessile

**Prognosis**—The prognosis is favorable in proportion to its early recognition and extirpation It is also favorable when the age of the patient exceeds twenty five years In other words small fibromas which do not fill the nasopharyngeal space are more favorable under operative treatment than those which completely fill it The tendency of the growth to undergo retrograde changes after the twenty fifth year accounts for the more favorable prognosis in those cases in which it occurs after this period Some patients recover spontaneously

**Treatment**—The various forms of treatment that have been used are caustics electrolysis galvanocautery snare avulsion in which the tumor is torn from its attachment by a rocking motion external operations and splitting of the palate Irradiation in the form of radium or the roentgen ray both pre and postoperative have given good results in most cases

Fibromas may be treated by embedding emanation seeds repeating the treatment five or six times at intervals of six or eight weeks if necessary Small growths especially if they are pedunculated and those limited to the nasopharyngeal space may be removed with a heavy snare either through the nose or mouth or with heavy forceps Electrocoagulation either direct or with a coagulating current through the snare has been used after the preliminary insertion of radium seeds

When the growth is large and sessile and has extensive inflammatory adhesions to the adjacent structures it may be necessary to perform an external or more radical operation



FIG. 312 — Fibroma removed from the nasopharynx Actual size (Specimen kindly loaned by A. G. W. Peters)

Large soft pedunculated fibromas may be removed as follows:

Prepare the patient as for a major operation. General anesthesia is preferable.

Place the patient in Rose's position. Be prepared to ligate the external carotid artery, and to introduce postnasal tampons.

Break down the inflammatory adhesions around the choanæ with the finger, which should be introduced through the mouth.

Introduce curved pharyngeal scissors (Fig. 55) through the mouth into the nasopharynx posteriorly to the body of the tumor until the pedicle of the tumor is reached, and sever it if possible. If the tumor is very large, this may not be possible.

If the pedicle cannot be severed with the scissors, introduce strong, slightly curved cutting forceps through the mouth into the vault of the nasopharynx, seize the pedicle, and cut it from its attachment to the base of the sphenoid bone. If the mass has a broad base and very hard, it may be grasped with the heavy Brandegee adenoid punch forceps and rocked back and forth until its attachment is torn loose.

In most instances requiring an external operation Moore's lateral rhinotomy will give sufficient exposure.

When the growth is so large that it invades the surrounding structures of the nose, and extensive adhesions are present, it may become necessary to resort to a temporary resection of the superior maxilla to eradicate it.

However, this operation is seldom required for the removal of a fibroma. It may be indicated in some cases of non-malignant fibrous or cavernous tumors which originate from the base of the skull, fill the nasopharyngeal space, and force themselves into the maxillary sinus, or through the sphenomaxillary fossa into the temporal fossa (retromaxillary tumors).

By reflecting a portion of the upper jaw upward, which has been sawn through, but which remains in connection with the soft parts, the tumor is completely exposed, so that it can be cut off from the base of the skull with a knife or scissors; this portion of the upper jaw is then replaced and the skin is sutured over it.

*Temporary Resection of the Superior Maxilla (Von Langenbeck)*—An external incision is made down to the bone in the form of a curve from the external angle of the nostril to the middle of the zygomatic arch (Fig. 313).

The insertion of the masseter muscle is separated from the lower margin of the malar bone portion of the buccal fascia.

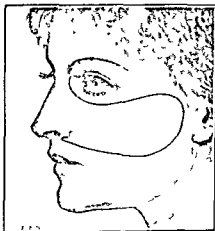


FIG. 313. Incision for the temporary resection of the superior maxilla.

After the lower jaw has been pressed downward by a gag inserted at the angle of the mouth on the healthy side the right index finger is forced into the sphenomaxillary fossa between the tumor and the upper jaw and then through the distended spheno-palatine foramen as far as the nares an elevator is carried along the finger, and on it a fine metacarpal saw is introduced into the pharynx. The left index finger, introduced from the mouth into the pharynx catches the point of the saw.

Horizontal division is obtained by sawing the upper jaw above the alveolar process as far as and into the pyriform aperture (Fig 314 b). In operations on the right upper jaw, the left index finger is forced into the maxillary fossa, and the operator saws toward it from the nasal passage.

The external incision is made down to the bone in the form of a curve from the upper portion of the nose (Fig 313) along the lower orbital margin, meeting the first skin incision at the zygomatic arch.

After the external lower angle of the orbit and the angle between the temporal and the frontal process of the malar bone have been freed from the soft parts, the zygomatic arch is sawn through in the middle from within outward next the frontal process of the malar bone as far as and into the inferior orbital fissure, the orbital plate of the upper jaw as far as the lacrimal bone closely below the lacrimal fossa and, finally, the middle of the nasal process of the upper jaw as far as the nasal bone is divided with a metacarpal saw. The organs which constitute the lacrimal duct should be protected.

By means of an elevator inserted under the malar bone the excised piece of the upper jaw is lifted up toward the median line like the lid of a box. The sutural connection between the nasal bone and the upper jaw in most cases breaks during this maneuver.

With a broad elevator the tumor, now laid bare, is lifted out of the sphenomaxillary fossa and the base is detached from the under surface of the skull with a knife, scissors, diathermy, or thermocautery. Finally the resected portion of the upper jaw is replaced in its former position and the skin is closed by sutures.

**Fibroma of the Tonsil**—**Etiology**—Fibroma of the tonsil occurs equally often in each sex and perhaps more often in the young than in



FIG. 314—Von Langenbeck's operation for the temporary excision of the superior maxilla. *a b* (Fig 310) the external skin incision. *c* the zygomatic arch is first sawn through from within outward. *d* next the frontal process of the malar bone is severed with a metacarpal saw as far as and into the inferior orbital fissure. the orbital plate of the inferior maxilla as far as the lacrimal bone closely below the lacrimal fossa and finally the middle of the nasal process of the superior maxilla as far as the nasal bones are divided. The contents of the lacrimal canal should be carefully guarded from injury. *b* horizontal division with a saw of the superior maxilla above the alveolar process as far as and into the pyriform aperture.

middle and advanced life. It is a benign neoplasm next in frequency of occurrence to papilloma. It very rarely becomes malignant. Its growth is very slow, and is usually limited to one tonsil.

**Pathology.**—The fibroma is usually somewhat pedunculated, though it may be sessile. The larger the fibroma, the larger the pedicle. It is more often single than multiple. Being of connective tissue of mesoblastic origin, it must of necessity have its origin from the trabeculae of the tonsil. Occasionally it undergoes cystic degeneration. Usually it is firm and scantily supplied with blood-vessels. It is composed of white fibrous tissue, the cells often being matted together, closely simulating embryonic connective-tissue cells.

**Symptoms.**—Annoying symptoms are seldom present except in the large pedunculated type, in which it produces mechanical obstruction. Its presence is not accompanied by discharge. It is characterized by symptoms similar to those of enlarged hyperplastic tonsils.

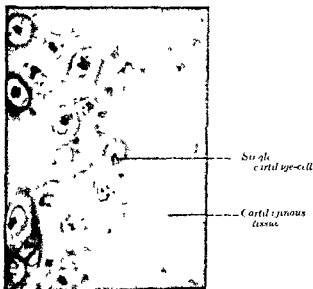


FIG. 315.—Chondroma (X 400). Cartilage is the basic tissue of a chondroma and differs microscopically from the normal cartilage in that the cells are arranged singly instead of in groups.

**Diagnosis.**—The diagnosis is usually easily made, and in case of doubt a portion should be excised and submitted to microscopic examination.

**Treatment.**—The treatment is purely surgical, and consists in its removal, a procedure easily accomplished if the growth is pedunculated. Occasionally it may be adherent to the tonsil or to the neighboring structures as a result of repeated inflammations of the tonsil.

**Surgical Technic.**—Anesthetize as for a tonsillectomy.

Separate the points of adhesion with a scalpel or scissors.

Pass a cold-wire snare around the tumor, engaging it at its pedicle,

or point of attachment. Sever the pedicle by closing the wire loop. If attached to the tonsil the tonsil should be removed.

Instead of using the wire snare the growth may be seized with the vulsellum or other toothed forceps and dissected with a scalpel from its attachment.

**Fibroma of the Larynx**—These growths occur more frequently in the male adult.

Their *etiology* probably has an inflammatory base. When pedunculated they resemble polyps both microscopically and macroscopically. The sessile type bears less resemblance. They are usually attached to the vocal cord. The pedunculated variety may appear or disappear with expiration or inspiration.

The *symptoms* are interference with the voice and rarely dysphagia. If the growth is large intermittent dyspnea may be present.

The *treatment* is removal by any method elected.

### CHONDROMA

Chondroma of the larynx is very rare. McCall, Dupertuis and Gardner<sup>1</sup> found a total of 55 cases including 2 of their own reported in the literature up to 1944.

Chondroma of the larynx may appear at any age but most frequently between forty and sixty years of age. Men are more frequently affected than women.

Most of the cartilaginous tumors of the larynx so far recorded have originated in one of the preexisting laryngeal cartilages. The growths usually arise from the cricoid or thyroid cartilages. According to Figi a small percentage originate from the epiglottis and arytenoid cartilages. Occasionally the growth occurs on the exterior of one of the laryngeal cartilages.

They are as a rule subglottic and may be overlooked when small unless the patient is examined in the upright position with the cords in the inspiratory position.

**Symptoms**—The symptoms may not appear for years after the inception of the growth. They are dependent on the size and position of the neoplasms. Hoarseness, dyspnea, cough and dysphagia are present according to whether the tumor is above or below the glottis. Stridor is usually a late symptom unless inflammatory changes occur.

Frequently the first change seen in the larynx is immobility of one vocal cord. The tumor usually appears pale, non-inflammatory, smooth and hard on palpitation with prominent blood vessels in an otherwise normal mucous membrane. The tumors are inactive and rarely ulcerate.

The diagnosis of chondroma of the larynx is made from the history, physical findings, roentgen ray examination and microscopic examination of the tissue.

<sup>1</sup> Laryngoscope 54:1 (January) 1944.

<sup>2</sup> Trans. Am. Laryngol. Assn. 1937.

**Treatment** — Removal by indirect direct or suspension laryngoscopy is done in cases in which the tumor is small and pedunculated or attached to the epiglottis otherwise a laryngofissure with enucleation of the growth with its capsule is the treatment of choice. A preliminary tracheotomy may be necessary especially if the tumor is producing respiratory obstruction. Incomplete removal has been followed by recurrence in some cases.

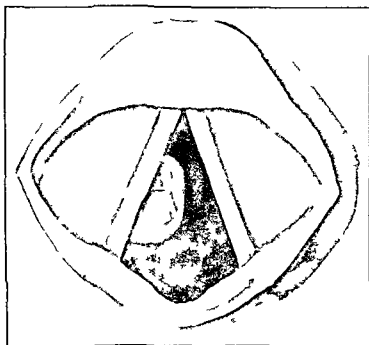


FIG. 316. Chondroma of the larynx originating from the right ala of the thyrocartilage. These growths grow slowly and as a rule produce gradually increasing symptoms of interference with respiration and deglutition.

**Chondroma of the Nose** — Chondroma involving the nasal structures is very rare. Very few cases have been reported. They are assumed to arise from cartilaginous rests. Chondroma of the ethmoid occurs chiefly between the ages of ten and twenty-five years (Hickey<sup>1</sup>).

Symptoms develop slowly and when present are orbital in character as a rule.

The treatment is surgical removal. There is a tendency to recur.

## ANGIOMA

**Etiology** — Angiomas of the nose, pharynx and larynx are rare. They are usually congenital.

The growths may not manifest themselves for years. The reported cases range from a few months to sixty years of age.

<sup>1</sup> Arch. Otolaryngol. 31: 645 (April) 1940.

**Pathology**—Angiomas are divided into hemangiomas and lymphangiomas

The hemangiomas are divided into four types the capillary or simple (telangiectatic angioma nevus etc) the cavernous the hypertrophic and the pseudoangioma (bleeding polyps fibroangiomas circoid aneurysms etc)

Capillary hemangioma consists of loosely arranged tissue containing numerous thin walled blood vessels showing at times areas of throm

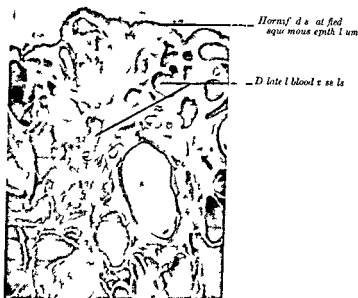


FIG 317—Hemangioma (X 30) Angiomas of the nose pharynx or larynx are rare and usually are congenital

bases hyaline degeneration and hemosiderin The tumor is covered with normal stratified epithelium Varices are usually found on the posterior wall of the pharynx or on the base of the tongue They frequently occur in association with chronic pulmonary or circulatory disturbances Slight hemoptysis may occur at times

Cavernous hemangiomas are thin walled with an afferent artery and efferent veins which do not communicate with the neighboring capillaries They may occur singly or in groups (Blair) Distended veins project above the surface and into the tissue beneath They may be compressed but refill at once when the pressure is removed Tumors of this type are usually slightly lobulated and dark purplish in color

Four types of lymphangiomata have been described The simple cavernous hypertrophic and cystic

The simple type (lymphangioma simplex) is characterized by a new growth of lymphatic vessels having a comparatively narrow lumen and more or less parallel walls

The cavernous type (lymphangioma cavernosum) is characterized by irregularly shaped intercommunicating lymph spaces of varying sizes

In the hypertrophic type (lymphangioma hypertrophicum) the lymphatic vessels are lined with several layers of endothelium with at times a nodular or valvular growth of endothelium into the lumen of the vessels

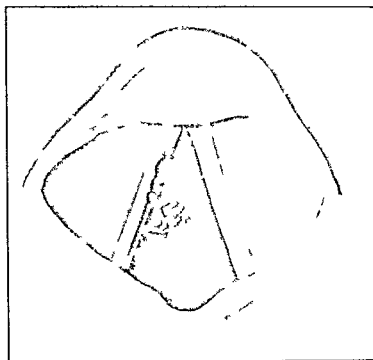


FIG. 318. Hemangioma of the larynx. Single hemangioma of the larynx may be nodular, polypoid, pedunculated or rare taes papillary. Lymphangiomas of the larynx usually have a smooth or papillary surface.

The cystic type (lymphangioma cysticum) is characterized by one or more large lymphatic cysts in a new growth of lymph vessels

Angiomas are sessile, non-ulcerating, with a nodular, serpentine outline and usually of a soft consistency. Hemangiomas have a purplish blue color. The lymphangiomas are smoother and pale pink.

A hemangioma may be associated with a similar process involving the skin. It is frequently found at the base of the tonsil or tongue arising from a venous plexus.

Cavernous hemangio-endothelioma of the nose is rare. It arises from the ethmoid region in the lateral wall. It grows slowly by infiltration and is destructive to surrounding tissues. It has a tendency to invade the sinuses especially in the region of the ethmoid, eventually filling the nose, sinuses or nasopharynx, producing in extreme cases exophthalmos, nasal obstruction or facial disfigurement.



Hemangioma of the nasal septum arises from the cartilage or cartilaginous vomer junction. It probably has its origin from embryonic rests.

Angiomas of the larynx, when associated with similar conditions elsewhere, may involve the extrinsic structures as well as the intrinsic. When localized in the larynx they are most frequently found in the true and false cords (New). Hemangiomas in the infant are rare but when present the reported cases have been subglottic.

**Symptoms**—The symptoms are variable, depending on the location and the size of the tumor. In some cases no symptoms are present until the tumor reaches a comparatively large size, in vocal cord lesion

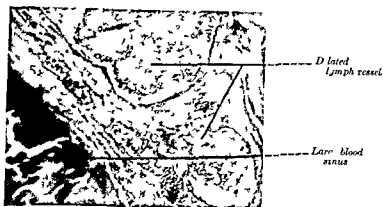


FIG. 319.—Lymphangioma ( $\times 80$ ). A lymphangioma is an angioma formed by dilated lymph vessels. It is rarely found in the ear, nose or throat.

attention is directed to the larynx by the voice changes such as hoarseness or aphonia.

If the tumor is large, slight or severe respiratory obstruction may be present. Hemoptysis may occur without warning and may be severe. Pain is usually absent. They may be pedunculated or more frequently they are attached by a broad base. They may be single or multiple. Lymphangiomas are usually smoother and paler than the hemangiomas.

The symptoms of angioma of the nose are those of more or less nasal obstruction, epistaxis, and a reducible and pulsating tumor. The nasal obstruction is proportionate to the size of the growth. Pressure upon the growth materially reduces its size. The pulsation is greater when the tumor is attached to a large artery than if it is attached to a vein. In the latter event the pulsation is much less and the color is blue, whereas if it is connected with both vein and artery the color will be dark red.

A microscopic examination is essential to an accurate diagnosis. There is no definite association with other general pathologic conditions.

**Treatment**—The treatment of angiomas is by roentgen ray, radium, cauterization, fulguration, strangulation and excision.

Roentgen ray has given excellent results in the treatment of this class of tumors.

Electrolysis is performed as follows: Anesthetize the tumor with a local application of a 10 per cent solution of cocaine, introduce the needles, connected with the positive pole of the galvanic battery, into the growth; turn on from 10 to 25 ma of current for five minutes. Repeat the seances at intervals of about seven days until the growth is obliterated.

The positive pole of the battery liberates nascent oxygen which coagulates the tissue, hence it is the pole which should be applied to a soft growth. If it is desired to reduce a hard or fibrous tumor, the negative pole is applied to the growth, because it liberates hydrogen, which softens the tissue.

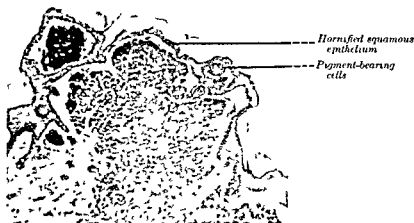


FIG. 320—Pigmented nevus of the face. Under the squamous epithelium of the skin collections of heavily staining pigment bearing cells are seen.

Ligation or strangulation may be performed as follows. Anesthetize the growth by the local application of a 10 per cent solution of cocaine, pass a ligature through the tissues including the artery at the margin or base of the angioma, and tie it. Yankauer's needles should be used, continue to tie off any remaining vessels until the nutrient sources are closed; after three or four days the ligatures should be removed.

The method of choice in treating all laryngeal angiomas is suspension laryngoscopy which permits the insertion of radium directly against the tumor.

Radical operations, such as thyrotomy with or without the cautery, should never be done except when all other methods fail to relieve the distressing symptoms, or in cases of large angiomas in which the danger of hemorrhage from removal is great.

In case an operation is decided upon in patients with large angiomas, thyrotomy with or without the cautery is preferable. It insures the best exposure and danger from hemorrhage is reduced to the minimum.

Treatment of angioma of the nose consists of radium, electrodesiccation, cauterization and excision. Excision seems to be the method of

choice. A simple resection including the cartilage leaving the submucous tissues on the opposite side prevents a perforation of the septum.

### HEREDITARY HEMORRHAGIC TELANGIECTASIA OSLER'S DISEASE

The type of angioma known as hereditary hemorrhagic telangiectasia is an inherited abnormality with a marked tendency to bleed.

**Etiology**—The disease is transmitted as a dominant characteristic and is not sex bound.

The disease is usually considered as a simple ectasia or dilatation of the blood-vessels but according to Stock<sup>1</sup> in his review of the literature is regarded by some as acquired multiple angiomas arising from embryonal endothelial rests.

The lesions may appear in various portions of the body but are most frequently found in the nasal or oral mucous membrane, especially the cartilaginous portion of the nasal septum, tongue, buccal regions, lips and the floor of the mouth. They are commonly observed in the skin of the face, scalp, tips of the fingers, eyelids, ear-drums, palate, pharynx, larynx and trachea.

The angiomas vary in size from a small pin head to large vascular networks and in color from red to purple. The tissue covering of the dilated vessels is extremely thin, permitting frequent hemorrhages.

**Symptoms**—Epistaxis or bleeding from other involved areas is the most common symptom. The bleeding varies from a mild loss of blood to a severe hemorrhage. It may occur at long intervals or several times a day. Secondary anemia is common.

Hereditary hemorrhagic telangiectasia should be differentiated from the various bleeding diseases and from telangiectases associated with other conditions such as syphilis, lead poisoning, senility, pregnancy, etc.

**Treatment**—Various means for destroying the angiomas or controlling the bleeding have been tried, such as irradiation, solid carbon dioxide, snake venom, vitamin K, injection of sclerosing solutions, electric cautery and electrocoagulation. Figg and Watkins<sup>2</sup> found electrocoagulation the treatment of choice. For the immediate control of epistaxis they insert a finger cot tied over the end of a catheter into the nostril and then inflate the rubber plug. Irradiation of lesions in the nasal mucosa is contraindicated in the opinion of most observers because of the secondary atrophy and crusting that follow. Transfusions of citrated blood may be necessary to combat the secondary anemia.

### NÆVUS

The word *nævus* is applied to two conditions, both innocent, the pigmented *nævus* or mole and the skin angioma or birth mark. Rarely the pigmented *nævus* may undergo malignant degeneration. Frequently *nævus* of the face is observed.

Microscopically a quiescent *nævus* consists of collections of clear

<sup>1</sup> Arch. Otolaryngol. 40: 108 (August) 1944.

<sup>2</sup> Ann. Otol. Rhinol. and Laryngol. 42: 330 (June) 1943.

rounded cells in the dermis often situated between downgrowths of epidermis. At the margin of these closely packed naevus cells are more fusiform pigmented cells filled with melanin granules these are melanoblasts. The tumor is of nervous tissue origin (Boyd).

### AMYLOID TUMORS

**Etiology**—Local amyloid disease of the upper air passages is relatively rare. Kramer and Som<sup>1</sup> found 95 instances of idiopathic local amyloid tumors up to 1934. The origin of these deposits is obscure. Men are afflicted more than women. The usual age of occurrence is between fifty and sixty years.

**Pathology**—Amyloid deposits in the mouth or larynx may occur secondarily within neoplasms or areas of chronic inflammation or as a part of a generalized amyloidosis. It occurs most frequently in the smaller arteries and capillaries especially in the media and intermediary layers of the intima. The upper air passages especially the larynx and trachea are involved more frequently than any other part of the body in a local amyloidosis. In rare instances they may occur as idiopathic or primary tumor like amyloid deposits. They may be single multiple or diffuse pedunculated or sessile usually the latter. As a rule a number of deposits are found extending to the cheek tongue tonsils larynx and trachea. They have a transparent wax appearance somewhat reddish or yellowish gray in color with an absence of ulceration lymph node involvement and pain.

**Symptoms**—Symptoms are those which accompany benign neoplasms of slow growth. Symptoms may be absent depending on the location. If in the larynx hoarseness or stridor may be noted depending on the size and location of the tumor. The rate of growth is very often slow usually requiring years before symptoms are evident. The diagnosis is difficult without biopsy however the intravenous injection of Congo red dye for detection of a generalized amyloid disease may be of help.

**Treatment**—The treatment of local amyloid deposits consists of excision where possible. Removal by direct or suspension laryngoscopy offers the best chance for a permanent cure. Laryngofissure may be necessary for complete removal of the mass. Radiotherapy has been found to be of value. Recurrences are common after removal.



FIG. 321 Amyloid tumors of the larynx

### CYSTS

**Cysts of the Mouth and Maxilla**—Common cysts of the oral cavity and maxilla include radicular or root cysts follicular or dentigerous

<sup>1</sup> Arch Otolaryngol 21:324 (March) 1935

cysts ranulas (retention cysts) hygromas and at times mucoid multilocular dermoid epidermoid and echinococcus cysts

**Root or Radicular Cyst** —The root or periosteal cyst arises from an inflammatory change in the root membrane due to injury or disease. It follows the death of the pulp with a subsequent development of a granuloma and a cyst. A roentgenogram shows an absence of a tooth in these cysts.

They comprise about 80 per cent of all dental cysts. They are found in the upper jaw more frequently than in the lower.

**Dentigerous Cyst** —*Etiology* —The follicular or dentigerous cyst comprises about 20 per cent of all dental cysts. It involves as a rule the lower jaw but may occur in the maxilla or even in the gums or orbit. It is usually found between twenty and thirty years of age but is occasionally seen in children or in old people. Most authorities are of the opinion that it represents an expanded tooth follicle. Magiot believes they originate from a periostitis at the root of a tooth. Malassez presents the theory that they originate from epithelial rests of the enamel germ. Dentigerous cysts are usually single but may be multiple.

*Pathology* —A dentigerous cyst should not be confused with a pathologic resorption of bone from an infection. An infection may spread by way of the root canal to the cyst. It is lined with pavement epithelium. They may vary in size from a small pea to a large walnut. The contents are sterile unless secondary infection occurs. They contain cholesterol crystals at times.

The cyst expands progressively with displacement and absorption of the bone so that the cortex may have the consistency of an egg-shell.

*Symptoms* —Symptoms may be absent until the walls of the maxillary sinus are beginning to be displaced. This displacement may be anteriorly into the palate or into the nasal passage. Destruction or perforation of the wall is a late development. Pain is absent as a rule.

An asymmetry of the face may occur consisting of a dilation of the anterior sinus wall or an inner displacement of the lateral nasal wall. The palate may be depressed at times by the expanding cyst. A fistula into the mouth or nose may occur if the cyst has ruptured. Transillumination may not show cloudiness of the antrum.

Examination may reveal a rounded swelling in the region of the canine fossa. A fistula in the mucous membrane of the oral or nasal cavities usually means an infection of the cyst. The cyst usually has a thin bony shell surrounding the contents which is easily stripped away from the wall of the antrum. The contents of the cysts may be a fully or partially developed tooth or teeth in a thin or gelatinous fluid which usually contains cholesterol crystals. In some instances multilocular dentigerous cysts are encountered (Love).

*Diagnosis* —The diagnosis is made from the history, roentgen ray and clinical findings. If a slow growing painless tumor of the lower jaw is found associated with an unerupted tooth, a dentigerous cyst should

be suspected. The roentgen ray reveals a large radiolucent area containing a tooth crown or an anomalous tooth.

Dentigerous cysts of the maxillary sinus should be differentiated from abscess, radicular or root cysts, osteomyelitis, maxillary sinusitis, benign tumors such as odontoma, fibroma, myxoma, etc. and malignant growths.

*Treatment*—Aspiration or incision and drainage of the contents is usually followed by a reformation or an infection of the cyst. The treatment of choice is a complete extirpation of the cyst and its bony wall by means of a Caldwell-Luc type of operation. If the cyst is infected, the bony shell may not separate from the intral wall easily. Following complete removal of the cyst, the prognosis is good.

**Ranula**—Ranula is a degenerative cyst formation of the salivary glands in the sublingual region. It is the result of a degenerative process of the salivary glands. A mechanical obstruction of the orifices of the duct is absent. It is usually located on one side of the frenum and has a tendency to recur.

*Treatment*—Aspiration or incision and drainage may not prevent a recurrence. A complete extirpation of the cyst is preferable either through the mouth or if the cyst is large and has burrowed inferiorly it may be removed through the neck.

**Hygromas**—Hygromas result from the occlusion of the ducts or orifices of mucous glands found in the region of the mouth. They are located in the mucous membrane while ranulae are beneath. Hygromas are true retention cysts lined with epithelium containing lymph structures. As a rule they are single but may be multiple and about the size of a pea. They may be located in any part of the mucosa of the mouth.

*Treatment*—The treatment consists in the enucleation of the cyst membrane though thorough cauterization of the lining of the sac is usually followed by the obliteration of the tumor.

**Multilocular Cyst (Cystic Adenoma or Adamantinoma)**—The multilocular cyst of the jaw bone is rare. According to Malassez it originates from epithelial rests of fetal life either from the mucous membrane of the jaw or from the epithelial cord or membrane of the enamel organ. It extends inward to the floor of the mouth from the lower jaw.

**Dermoid Cyst**—A dermoid cyst in the floor of the mouth develops from a misplaced fetal inclusion that takes on activity and forms a cyst.

Dermoids commence shortly after birth but on account of their slow and painless growth attract little attention until their size gives annoyance. They project into the floor of the mouth or into the neck behind the chin or both places. At times they attain the size of an orange.

The round or oval dermoid cyst has a thick wall lined with stratified epithelium. It usually contains hair or other skin appendages and certain fatty bodies and fatty acid crystals.

Dermoid cysts of the dorsum of the nose are very rare. Hagens<sup>1</sup> in

1938 found 26 cases reported in the literature. They are probably present at birth but do not become manifest as a rule until from ten to twenty years of age. The base or stalk of the dermoid usually arises from the nasal or frontal bones but the cyst may present itself at the glabella, along the dorsum or at the tip of the nose (Holmes<sup>1</sup>).

Treatment is by the complete surgical removal of the cyst and its stalk.

**Epidermoid Cyst**—Epidermoid cysts are similar to dermoids as they both come from the same embryonal structures. However the epidermoids do not contain hair glands or skin appendages. The epidermoids are lined with an epithelial layer of a mucous type and sometimes with ciliated epithelium (Johnston<sup>2</sup>).

**Mucous Gland Cysts**—These retention cysts may form in any mucous gland in the mouth except the gingiva and superior surface of the tongue. The duct or its orifice becomes occluded causing the formation of the pseudocyst. They may be mistaken for a ranula.

**Echinococcus Cyst**—The echinococcus cysts are rarely found in the mouth. When present they are usually located on the tongue. Pain is absent as a rule unless a secondary infection occurs. A mechanical inconvenience may be noticed from the presence of the cyst.

**Cyst of the Tonsil**—Cysts of the tonsil are rare. They may be either superficially or deeply situated. They vary in size and may contain a quantity of fluid or a mass of inspissated secretions and epithelial debris. Cysts usually result from an inflammatory occlusion of the mouth of the crypt.

**Cysts of the Nasopharynx**—Small retention cysts are common in the palatine tonsils. They may form from an obstructed and dilated crypt.

Cysts of the nasopharynx may be inflammatory, degenerative or developmental in origin. It is possible that many of the larger cysts of the nasopharynx lined with ciliated epithelium originate as cysts of the pharyngeal bursa (page 310).

Subjective symptoms referable to the nose or throat may be absent. Postnasal discharge is a common symptom. Chronic pharyngitis and enlargement of the posterior cervical lymph nodes are present frequently. Occipital headaches are mentioned by some patients.

Diagnosis is made by direct inspection and palpation of the nasopharynx. A smooth bulging in the vault of the nasopharynx usually central but occasionally lateral is usually seen.

**Treatment**—The treatment is destruction with the actual cautery or surgical removal. The technic is similar to that followed in the removal of an adenoid.

**Cystadenoma**—Cystadenoma of the palate is rare. New<sup>3</sup> in 1916 found a total of 46 cases including 2 of his own. They are thought to be embryonic or to originate from the germinative layer of the epithe-

Ann Otol Rhinol and Laryngol 51 662 (September) 1942  
<sup>1</sup> Ann Otol Rhinol and Laryngol 51 917 (December) 1942  
<sup>2</sup> Ann Otol Rhinol and Laryngol 25 687 (September) 1916

hum of the palate. The tumors are usually discovered accidentally. As a rule the hard palate is involved but the soft palate and the gums may be the seat of the cystic tumor.

They are irregularly oval or rounded sessile growths, grayish or pinkish-gray in color and soft or firm in consistency. They usually

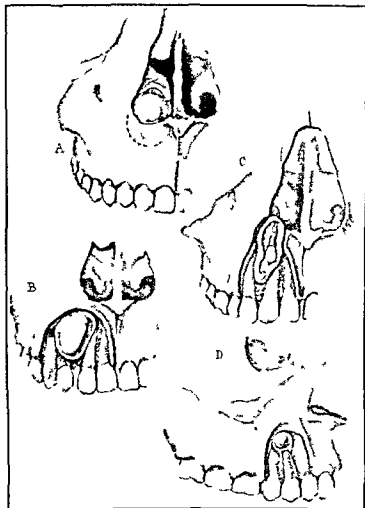


FIG. 377.—Naso-alveolar cysts. *A* A naso-alveolar (anterior) cyst situated anterior to the lateral incisor and canine teeth. *B* A globulomaxillary cyst situated between the lateral incisor and canine teeth. *C* A dentigerous cyst containing an anomalous tooth. *D* A dental root cyst, also known as a dentoperiosteal or radicular cyst, may be found at the apex of any tooth root.

are encapsulated, but may break through the capsule. A glutinous colloid material, glandular epithelium and at times cartilage, bone and lymphoid tissue may be found within the capsule. The cystic formation is due to the dilated acini.



**Treatment**—Treatment is by excision with the knife or cautery followed by cauterization of the base

**Cystadenoma of the Larynx** The numerous mucous glands in the larynx may give rise to an adenoma which may undergo cystic degeneration. This type of tumor is rarely seen in the larynx however

Cystadenoma of the larynx is more common in men than in women and all the reported cases have been in adults (Figi and Rowland). It is usually situated in the ventricles, ventricular bands, epiglottis or subglottic regions

The cysts lined with a single layer of cylindric epithelium at times filled contain clear mucus or a yellowish sebum like fluid

Hoarseness is the most common symptom. Cough is infrequent. If the tumor is large dyspnea may occur

Examination usually reveals a single sessile growth but at times a pedunculated type or bilateral growths may be observed

Cystadenoma of the larynx should be differentiated from prolapse of the ventricle or sacculus, polyp, various granulomatous lesions and malignant growths

Treatment is the surgical removal by direct or indirect laryngoscopy with small growths and by laryngofissure in large ones. According to Figi and Rowland suspension laryngoscopy is the method of choice. Electrocoagulation may be used for searing the attachment after the surgical removal

**Cysts of the Nasal Vestibule and Hard Palate**—Cysts encroaching on the nasal vestibule or the anterior portion of the hard palate are rare. They are usually unilateral and largely confined to the female sex. These cysts formerly assumed to be mucoid are thought to be due in most instances to a persistence of epithelial remnants in the line of closure of the premaxilla and the maxilla (facial cleft cyst) or in the line of fusion of the palatal processes (incisive canal cyst, nasopalatine duct cyst)

**Naso alveolar Cyst** is the most common type derived from the facial cleft. This extra-osseous type of cyst situated on the bone below the piriform aperture may elevate the floor of the nasal vestibule or even cause an asymmetry or distortion of the features (Rosenberger<sup>1</sup>)

Symptoms are varying degrees of nasal obstruction on the affected side depending upon the size of the cyst

Examination reveals a smooth round mucosa-covered cyst in the vestibule of the nose beneath the anterior tip of the inferior turbinate. Palpation beneath the upper lip may reveal the lower margin of the fluctuant cyst

Treatment is by surgical removal through a medially placed Caldwell Luc incision

**Globulomaxillary Cyst** is similar to the naso-alveolar type except it is posterior to the latter as the globulomaxillary cyst arises from epithelial

rests situated between the globular and maxillary processes rather than anterior to these processes (Sood<sup>1</sup>)

These cysts are located between the canine and lateral incisor teeth and usually produce their separation. The roentgen ray reveals an oval or heart shaped area between the apices of these teeth. Large cysts may result in sufficient thinning of the maxillary cortex to produce crepitation.

The treatment varies from simple incision and drainage to a radical removal of the cyst from the palatal side with or without the adjacent teeth.

**Nasopalatine Canal or Duct Cysts** produce a mid line swelling on the anterior portion of the hard palate. If large enough mastication may be difficult.

**Branchial and Thyroglossal Cysts and Fistulas**—Included among the various developmental anomalies of the neck are branchial cysts and fistulas and thyroglossal cysts and fistulas.

Branchial cysts or fistulas are thought to be due in most instances to remains of the thymic duct which descends from the third pharyngeal pouch (Wenglowski) especially the lateral cysts or fistulas below the level of the hyoid. True branchial cysts or fistulas are above this level (Meyer<sup>2</sup>). From a clinical standpoint the two types may be considered the same. These lateral defects of the branchial or thyroglossal systems may be located at any level of the neck. The tract extends below the anterior portion of the sternomastoid muscle and anterior to the carotid sheath to the posterior belly of the digastric muscle arches behind the stylopharyngeus muscle and ends in the tonsillar fossa. The tract is lined with ciliated epithelium and some layers of squamous epithelium.

Defects of the branchial tract are usually found in young people and predominantly in females.

The usual symptoms are a tumor in the lateral portion of the neck or a fistula with an intermittent or continuous discharge of secretion. Recurrent attacks of inflammation are common. An unexplained cough may be present at times. Symptoms may be noticed from a few days only to many years.

Thyroglossal cysts may form anywhere in the mid line of the neck along the tract of the vestigial thyroglossal duct from the base of the tongue to the region between the hyoid bone and the thyroid gland. Most of these retention cysts occur below the level of the hyoid bone. They may vary in size from a barely perceptible tumor to the size of a grapefruit.

Thyroglossal fistulas open on to the surface of the neck in the mid line or more rarely in the foramen cecum above the dorsum of the tongue. They usually develop from an infection or inflammation of a cyst or

Laryngoscope 54:18 (January) 1944

<sup>1</sup> Arch f klin Chir 93:151 1912

<sup>2</sup> Arch Surg 35:766 (October) 1937

the duct itself. The fistulas discharge a glairy mucus either clear or milky in color. If the tract is infected the discharge is purulent.

Symptoms are those of a mid line tumor or discharging fistula which moves upon swallowing. Difficulty in swallowing or a choking sensation may be present but they are rare. A secondary infection of the cyst or thyroglossal tract is common.

The diagnosis of branchial or thyroglossal cysts or fistulas is facilitated by injecting a radiopaque oil in the suspected tract after closing the opening with a purse string suture and then having a roentgenogram taken.

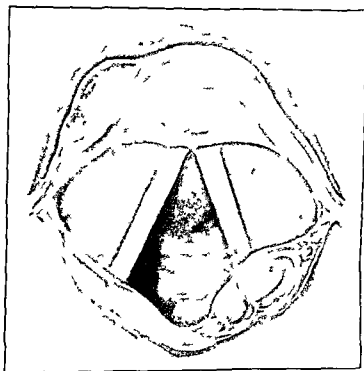


FIG. 393.—Medial cysts of the epiglottis and left arytenoid region.

The treatment of the fistulas or cysts by the injection of sclerosing solutions has been very unsatisfactory.

The complete surgical extirpation of the cyst or fistula gives the best assurance of a certain cure. The tract of the fistula can be followed more easily by a preliminary injection of a methylene blue solution. The entire tract from the neck to the foramen cecum (thyroglossal) or from the neck to the pharynx (branchial) must be removed. It may be necessary to resect the central portion of the hyoid bone when removing a thyroglossal cyst or fistula.

**Cysts of the Larynx — Etiology** — Cysts of the larynx are rare. They may be divided into four groups: retention cysts, blood or lymph cysts, parasitic cysts, and embryonal or congenital cysts.

The retention cyst is the most common. It is usually small and may be located in any part of the larynx, but is found most frequently on the lingual surface of the epiglottis and in the ventricle where mucous glands are numerous. They are formed by the closing of the duct of a gland with obstruction to the outflow. The retaining sac created forms the cyst.

The blood or lymph cysts are due to extravasation of blood or lymph in the subepithelial tissue. They are found usually about the vocal cords.

Laryngeal cysts due to echinococcus are rare but may occur.

Congenital or embryonal cysts may be found at any age. They are usually located on the aryepiglottic folds and on the lateral laryngeal wall. They contain both endodermal and mesodermal structures.

**Pathology** — A lining membrane of squamous cuboidal or columnar epithelium may be present in laryngeal cysts. The cysts may be round or oval, sessile (usually) or pedunculated. They may vary in size from that of a pinhead to a large egg. Smaller cysts are frequently found on the vocal cords, while the larger ones occur on the epiglottis. The majority of epiglottic cysts are located on the lingual aspect.

The small sessile laryngeal cysts, usually found on the vocal cords, contain serum, blood, milk, or cheesy material due to retention from the glands of the normal epithelium.

**Symptoms** — The symptoms vary greatly depending upon the size and location of the cyst. In many instances symptoms are absent. In others clearing of the throat, a change in voice, hoarseness or cough are present. If the cyst is large, cyanosis, dysphagia, stridulous breathing or dyspnea may be observed.

**Diagnosis** — The diagnosis is made from the symptoms and by the direct mirror or digital examination. A roentgenogram may be of value.

The differential diagnosis should be made from a prolapse of the ventricle, papilloma, diphtheria, asthma or foreign body.

The prognosis may be grave if the cyst is of large size or suddenly increases in size.

**Treatment** — The treatment varies according to the size, location and age of the patient. A simple incision or a cautery puncture may relieve the symptoms. If the fluid reaccumulates, removal of the cyst by a direct laryngoscopy is indicated. In some instances a thyrotomy and excision may be necessary.

If pedunculated, a snare may be used to remove the cyst and the stump cauterized with a cautery tip by the indirect or direct method.

**Laryngeal Cysts in Infants** — Laryngeal cysts in the new born are very rare. Kleinfield<sup>1</sup> states about 15 cases have been reported in the literature in which 7 were found at autopsy.

<sup>1</sup> Arch Otolaryngol 19:590 (May) 1934

The etiology is somewhat obscure. They are probably congenital anomalies rather than retention cysts. As a rule they have a thin wall which contains a milky fluid. They usually originate in the region of the laryngeal aperture.

The symptoms are those of obstruction such as dyspnea and cyanosis. Changes in the voice such as stridor or aphonia may be present.

The diagnosis is made from the history, roentgenograms and direct laryngoscopy.

The differential diagnosis should be made from congenital prolapse of the larynx, papilloma, foreign body, atelectasis, thymic enlargement and patent foramen ovale.

Incision of the cyst by direct laryngoscopy may be effective.

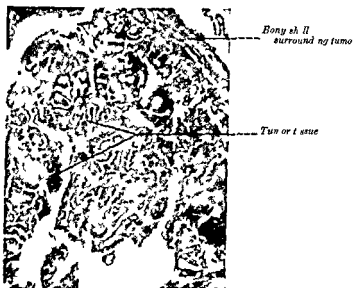


FIG. 324 — Adamantinoma ( $\times 30$ )

### ADAMANTINOMA—ODONTOMA—MULTILOCULAR CYST

An adamantinoma is a tumor which arises from the paradental epithelial debris. It is solid or cystic, located most commonly in the mandible in the molar and incuspid region and at times in the maxilla. It excavates a cavity and destroys the tissue until the tumor is surrounded by only a thin capsule which crepitates on pressure.

The adamantinoma in the early stage is solid but cystic degeneration appears later and gives them a softer consistency. They are composed of epithelium growing in masses or cords resembling embryonic enamel organs. Microscopically enameloblasts at some stage of development usually are visible, most often lining the cavities in the bone.

The slow growing tumor has a tendency to grow uninterruptedly, recur if incompletely removed and give rise to metastasis (Havens<sup>1</sup>)

The outstanding symptom is the slowly enlarging growth of the jaw. Slight pain may be mentioned in advanced cases.

Included in this group of tumors in addition to the odontomas are the dentigerous cysts which are described separately.

The hard odontoma, a tumor of dental origin, is composed of enamel, cement or dentin. Two or more of these tissues may be included in the same tumor.

The hard odontomas grow by direct expansion, while the adamantinomas grow by expansion and infiltration. Metastases do not occur in either type.

Odontoma of the maxillary antrum is comparatively rare but if invaded a facial deformity may result.

These tumors should be differentiated from a dentigerous cyst and osteosarcoma. The dentigerous cyst is slow-growing and painless. The roentgenograms show round smooth shadows with definite outline. The odontoma is dense and hard. Osteosarcoma will show signs of malignancy. Bone cysts in the early stage may be mistaken for an odontoma. In the latter the fluid content may be demonstrated if the wall is thin.

The treatment is by radical surgical or diathermic removal.

### MULTIPLE MYELOMA

Multiple myeloma appears to be a malignant type of tumor formation of the bone-marrow which rarely metastasizes. It is usually seen in the fourth or fifth decade of life.

Cases have been reported in which skeletal lesions were associated with involvement of the tonsil, lower jaw, cheek and pharynx.

The urine in 80 per cent of the cases shows the "Bence-Jones protein". The blood is usually normal except for the secondary anemia that may occur. The patient complains of neuralgic pains and later painful swellings of the involved areas of the skull or elsewhere.

A lymphoid or plasma cell myeloma and a myeloid myeloma have been described.

Radium or roentgen rays may give a temporary cure. Death usually occurs in from a few months to a few years.

### PLASMOCYTOMA

**Synonyms**—Myeloma, plasmosarcoma, malignant plasmoma.

**Etiology**—Plasmocytoma, occurring in the upper respiratory tract, is a comparatively rare form of myeloma resembling histologically the multiple myelomas commonly found in the long bones.

The etiologic nature of the tumors is still to be determined. They are most frequently found in association with chronic inflammatory conditions such as syphilis and tuberculosis.

<sup>1</sup> Arch. Otolaryngol. 31:762 (November) 1939.

Their degree of malignancy varies some of the reported cases being highly malignant while others appear to have run a benign course

**Pathology** The growths occur chiefly in the nasopharynx alveolar borders tongue lips and cervical lymph nodes but they have been observed in many other regions

The plasma cell granuloma usually shows a plasma cell infiltration or proliferation a marked response of the reticulum cells and at times the presence of eosinophils

**Symptoms** The predominant symptoms are nasal obstruction difficulty in deglutination epistaxis and hoarseness The course is relatively slow

**Prognosis**—Multiple myelomas are fatal as a rule However the solitary plasmocytoma is more amenable to treatment

**Treatment**—A combination of surgery and roentgen ray or radium seems to give the best results

### LEUKEMIC TUMORS (SEE LEUKEMIA)

In chronic lymphatic leukemia a leukemic infiltration of the mucosa submucosa and around the mucous glands of the nose or throat may occur producing nodules which may ulcerate The tonsils may be the seat of the lymphoid enlargement Aural complications are cellular leukemic infiltration and hemorrhage

### MENINGIOMA

A meningioma (olfactory groove meningioma) is derived from and attached to the dura which covers the cribriform plate of the ethmoid The tumor develops from the leptomeningeal cells which line the arachnoid villi

Elsberg<sup>1</sup> found meningiomas of the cribriform occurred in 1.2 per cent of a series of 1204 tumors of the brain and in 7.7 per cent of 195 intracranial meningeal growths

It may involve the olfactory nerves very early and disturb the sense of smell Later the optic nerves or chiasm may be involved with resulting diminution of vision

If the growths are large there is a history of impaired vision loss of smell and certain mental and neurologic disturbances

Meningiomas involving the temporal bone have been reported (Risch<sup>2</sup>) The reported cases have been found in women and men in the ratio of about 3 to 2 They usually appear during the third or fourth decade of life Extensive bone destruction without a productive bone reaction is characteristic The labyrinth is seldom involved

An early symptom frequently present is pain in the temporomandibular joint The meningioma presenting itself as a polypoid mass in the external auditory canal may bleed freely

Treatment is by deep roentgen ray therapy or radium

<sup>1</sup> Laryng 45 712 (September) 1935

<sup>2</sup> Laryngoscope 52 737 (September) 1942

## MENINGOCELE MENINGO-ENCEPHALOCELE

A meningocele is a hernial protrusion of the meninges. It may extend intranasally, but is rarely seen. Hallemann<sup>1</sup> found very few cases reported in the literature. The hernia opening is in the region of the lamina cribrosa. The hernia may push the mucous membrane of the roof of the olfactory fissure and the nasal septum before it, forcing the structures aside. If brain substance is enclosed within the meningocele the hernial formation is called a meningo-encephalocele.

Rhinoscopy reveals a tumor like formation covered with mucous membrane, which more or less fills the interior of the nose, continuous with the mucous membrane of the septum. The tumor is elastic with pulsation at times. Compression of both jugular veins results in a loss of elasticity.

A meningocele should be differentiated from polyps (usually movable), fibromas (tougher consistency and free bleeding), and malignant tumors. An exploratory puncture should be done cautiously as it may produce a fatal meningitis.

The surgical approach for removal of the intranasal protrusion may be made by a somewhat similar technic as that described for cerebrospinal rhinorrhea. If the pedicle is small an external ethmoid sinus approach similar to the fronto-ethmoid-sphenoid operation might be feasible.

## TERATOMA

Tumors and cysts of teratologic origin the result of congenital disturbances of development or misplacement of embryonal cells, have been observed in the nose, pharynx, mouth and neck.

Furstenberg<sup>2</sup> classifies these tumors and cysts into (1) those due to disturbances of the single individual embryo and (2) those due to disturbances of the twin embryo. The classification would include disturbances of dentition, branchiogenic, craniopharyngeal duct, irregularities of the thyroglossal duct and neurogenic disturbances.

Those which involve the twin embryo are

- 1 Cysts in the floor of the mouth, submaxillary glands, the mucous glands in the mucous membrane of the floor of the mouth and some irregularity in the closure of the second branchial cleft. For all these cysts the term "ranula" is used. Some of them however are acquired that is, due to closure of the ducts of mucous glands.

- 2 The lingual goiter or aberrant thyroid tissue.

- 3 Tumors involving the deep peripheral nerves called "perineurial fibroblastoma," "perineurial fibroma," "solitary neurofibroma," "peripheral glioma" and "schwannoma."

- 4 Tumors derived from the brain due to some disturbance in the embryonic development such as (a) a pinching off within the nasal cavity of a part of the primitive bud, forming a glioma in the nose,

<sup>1</sup> *Ztschr f Hals- Nasen u Ohrenh* 30 413 (May 31) 1932

<sup>2</sup> *Arch Otolaryngol* 24 406 (September) 1936



(b) the formation of a meningocele or meningo-encephalocele from a dehiscence in the region of the cribriform plate

5 A chondroma developing from cartilaginous cells which fail to undergo ossification. A chondrosarcoma may develop from these cartilaginous cell rests. It has been found in the nose, septum and various other parts of the head.

6 Mixed salivary tumors

7 The large embryoma is a type of neoplasm affecting two or more layers of the body. It is prone to occur in the pharynx and in the neck. It contains a profusion of connective tissue elements from the different germinal layers of the body.

These various tumors are described separately elsewhere; therefore further consideration will not be given here.

### NEUROFIBROMA

Neurofibromas developing about the face and neck are rare. When present they usually occur in association with generalized neurofibromatosis (von Recklinghausen's disease). Solitary neurofibromas may develop in the submaxillary region and secondarily bulge into the floor of the mouth or the base of the tongue. If they arise in the region of the neck they may in time encroach on the pharynx. Igit<sup>1</sup> reports a case of solitary neurofibroma primary in the pharynx.

Neurofibroma of the larynx is very rare. The usual location of the reported cases have been in the regions of the ventricular bands and aryepiglottic folds (Smith<sup>2</sup>).

The symptoms of hoarseness, cough, slight discomfort and at times dyspnea are those of any benign growth of the larynx depending upon the size and location of the tumor.

Neurofibroma of the larynx appears upon examination to be that of a gray or yellowish red, firm, somewhat round, encapsulated growth.

Treatment is by surgical incision.

The schwannomas arise from the sheath or from the nerve roots. The neurofibromas are found peripherally as a rule. They may arise from the root of the eighth cranial nerve (see cerebellopontine angle tumors) or from the root of the spinal nerves. They occur at times on other cranial nerves, especially the optic and trigeminal.

The tumors are rounded or nodular, encapsulated and may be hard or cystic.

### SCHWANNOMA—NEURINOMA

Schwannoma of the larynx is rare. It is formed by elements of the Schwannian syncytium, the delicate protoplasmic envelope enclosing every internodal segment of the adult medullated nerve fiber (Vail<sup>3</sup>).

The tumors frequently arise from the aryepiglottic fold. They may

<sup>1</sup> Arch. Otolaryngol. 17: 386 (March) 1933.

<sup>2</sup> Arch. Otolaryngol. 39: 144 (February) 1944.

<sup>3</sup> Trans. Am. Laryngol., Rhinol. and Otol. Soc. ety. 1933.

be pedunculated or not. The tumors frequently grow to be of large size, almost filling the larynx.

The symptoms are those of gradually increasing laryngeal obstruction to breathing and swallowing. Examination usually reveals a smooth, hard, rounded mucous membrane covered tumor. Roentgen ray examination may reveal the extent and size of the growth.

The surgical removal of the large non-pedunculated tumors usually is by external approach through the neck. Paralysis of a vocal cord has been reported in a number of cases following removal by the external route. In Vail's case the tumor was removed under suspension laryngoscopy with a successful outcome.



*Characteristic whorl  
of tumor cells*

FIG 325 —Neurofibroma ( $\times 30$ ) A neurofibroma is usually a benign tumor of rather complicated origin. In most instances an overgrowth of both the connective tissue elements of the nerve (epineurium, perineurium, and endoneurium) and the specialized nerve structures is seen.

Schwannomas of the tongue have been reported (Gnassi and Barone<sup>1</sup>). The tumor may be located in any portion but 4 of the 10 reported cases have been on the side. The tumor mass is usually soft and movable.

Symptoms are absent other than interference with the function of the tongue.

Treatment is by excision of the growth with its capsule.

Schwannomas of the facial nerve have been reported in recent years. Roberts<sup>2</sup> in 1943 found 13 cases including his own.

Symptoms of the slow growing tumor are from pressure on the facial nerve such as facial paralysis, nerve type deafness and possibly dis-

<sup>1</sup> Arch Otolaryngol, 27, 766, (June) 1938

<sup>2</sup> Arch Otolaryngol, 37, 62, (January) 1943

turbance of taste in the anterior two-thirds of the tongue on the involved side. If the middle ear is invaded a conduction type of deafness may ensue. Labyrinthine disturbances may occur from direct invasion of the labyrinth or from the secondary labyrinthitis.

### MYOBLASTOMA

Myoblastomas of the larynx are very rare. They may occur wherever striated muscle is found. Myoblasts, the ancestral cells of striated muscle, are essential components of the tumors. According to Kleinfield<sup>1</sup> these tumors are composed of two types of cells: a polygonal cell with granular cytoplasm (resembling early embryonal muscle fiber). Some of these tumors, especially those involving the vocal cords, often show hyperplasia of the overlying epithelium, thus simulating carcinoma from which they can be differentiated by the scarcity of mitosis or atypical cells. The tumors differ from xanthoma microscopically by the absence of fat, as shown by failure to stain with sudan. They are probably due to abnormal histogenic development, possibly following trauma, rather than to degeneration of preexisting striated muscle fiber. These growths have been found in persons of any age, but mostly in middle-aged men. (A form of myoblastoma is found in the newborn, so-called congenital epulis.) The tumors have a predilection for the upper respiratory and digestive tracts. Clinically they are benign.

### CHORDOMA

A chordoma, a neoplasm of the notochord, arises from the embryonic remains of the *chorda dorsalis*. They may be produced at any point along the axial skeleton where the notochordal cells are found. They are classified as cranial, vertebral and sacrococcygeal. Fifty-two cases have been reported as occurring in the spheno-occipital region (Gould).

They occur with greater frequency in males, especially in the third, fourth and fifth decades.

The symptoms of chordoma in this region are due to disturbed function of the cranial nerves or to compression of the brain stem or spinal cord. They are slow growing as a rule.

Chordoma projecting into the nasopharynx may be seen by the nasopharyngoscope and may be palpated. They may be mistaken for sarcoma or fibroma or other smooth, round, firm growths which erode the skull into the nasopharynx and involve the cranial nerves.

The prognosis is poor, depending on the involvement of the brain and spinal cord.

Surgical intervention may be successful occasionally, but is followed usually by a recurrence. Roentgen or radium therapy is sometimes palliative. The diagnosis is made by biopsy.

## RHABDOMYOMA

Rhabdomyoma involving the upper respiratory tract is very rare. Any muscular region in the body may show an occasional development of a rhabdomyoma. According to Ewing<sup>1</sup> the neck and adjoining region is a common location for the growths. Cases have been reported in the nose, orbit, tongue, esophagus and parotid gland. Cooper<sup>2</sup> reports a case of rhabdomyoma of the paranasal sinuses.

It is thought that growths originate from displaced embryonal cells. Some authors believe these tumors benign at their onset may become malignant. Metastasis takes place via the lymphatic lymph nodes. The cross striations common to these tumors are found in the lymph node metastases (Cooper). Cartilages, bone and other tissues have been found in the tumors.

Spindle-cell sarcoma, myosarcoma or tumors associated with anaplastic changes should be differentiated.

Surgical removal combined with postoperative irradiation is the best method of treatment.

## LINGUAL THYROID—ACCESSORY THYROID

**Etiology**—A lingual thyroid may result from an arrested descent of the thyroid anlage (heterotopic). In this form the cervical thyroid would be absent. Another form develops from aberrant rests (accessory thyroid, aberrant thyroid). The thyroid tissue is located in the great majority of cases in or on the base of the tongue between the epiglottis and the circumvallate papillae or at the site of the foramen cecum. A small number extend into the tongue above the frenum. They have their origin from the suprathyroid region. A sublingual type may be seen in the submental region as a rounded swelling beneath the skin.

It is found much more common in women, especially during periods of endocrine activity.

**Pathology**—They vary in size from very small tumors to that of a hen's egg. The usual size is about 2 by 2 cm. in width and length and 2 or 3 cm. above the base of the tongue.

**Symptoms**—Symptoms if present of a lingual thyroid are due to the enlargement of the tongue. A sense of foreign body, dysphagia or dysarthria are usually mentioned. If large, dyspnea and asphyxia may be present. Hemorrhage due to ulceration or rupture of a vein may occur.

On examination a circumscribed red swelling on the base of the tongue, sessile or pedunculated, at times with dilated vessels can be seen. The color is usually reddish or bluish. They may be semisoft or firm. It is painless and does not blanch with pressure.

<sup>1</sup>Neoplastic Diseases, Ed. 3, 1928.

<sup>2</sup>Arch. Otolaryngol. 20:379 (September) 1934.

Histologic examination usually reveals normal thyroid tissue with at times some changes of a colloid or parenchymatous nature

**Differential Diagnosis**—Lingual thyroid tumor should be differentiated from other benign tumors such as angioma amyloid tumors cysts, fibroma, papilloma, gummata, hyperplasias of the lingual tonsil, lipoma, epithelioma and lymphosarcoma. The diagnosis is proved by histologic examination of an incised portion

**Treatment**—Symptomless intralingual thyroid does not need to be removed. The presence or absence of other thyroid tissue in the normal location should be determined before removing the lingual growth as the removal of the lingual thyroid has been followed by myxedema and tetany

Surgical intervention should be reserved for those cases presenting dysphagia and dyspnea. Surgical diathermy with an electrocoagulating current has been used in recent years for removing enough of the gland to relieve the mechanical obstruction

External operations such as a lateral pharyngotomy by either the suprahoid or subhyoid routes have been employed in extreme cases

### CAROTID BODY TUMOR

Tumors of the carotid body are rare. Violet<sup>1</sup> states some 200 cases have been reported in the past fifty years

**Etiology**—The cause is unknown. It may occur at any age and in both sexes equally. A number of familial cases have been reported

**Pathology**—According to Twing<sup>2</sup> a carotid body tumor is a type of alveolar perithelioma but differing in some details. The firmly attached solid growth is usually unilateral and grows at the bifurcation of the carotid. The tumor is smooth, round or oval, and slightly compressible. According to Gratiot,<sup>3</sup> from 15 to 20 per cent become locally malignant in a late stage

**Symptoms**—The slowly developing tumor is usually symptom free for years except for the external swelling below and posterior to the angle of the jaw. In a later stage pressure of the growth may cause hoarseness, cough, tinnitus, nausea or possibly localized pain

The differential diagnosis should be made from the various types of lymph node enlargements, branchial clefts, aberrant thyroid, lipoma and malignant growths

**Treatment**—Treatment is by surgical excision. Ligation of the common carotid may be necessary

### BENIGN GIANT CELL TUMOR

The benign giant cell tumor is a low grade, neoplastic growth, usually single, affecting the epiphysis of the long bones as a rule but cases have been reported in which the maxilla, ethmoid, sphenoid, temporal and

<sup>1</sup> Ann Otol Rhinol and Laryngol 53:569 (September) 1944

<sup>2</sup> Neoplastic Diseases 1940

<sup>3</sup> Surg Gynec and Obst 77:177 1943

frontal bones have been involved in that order of frequency. Wattles<sup>1</sup> in a review of the literature found seven cases of giant cell tumor of the ethmoid.

The cause of a giant cell tumor is unknown. Among other theories it has been attributed to trauma or inflammation of the bone. It is more common in females and usually appears in the second or third decades of life.

The course is progressive, however, spontaneous cures are sometimes observed. Metastasis does not occur. Death is usually from hemorrhage or secondary infection. Most cases can be controlled by irradiation therapy.

### MIXED TUMOR

Mixed tumors, usually found in the parotid gland, may occur in some instances in various parts of the nose and the throat such as the palate, tonsil, pharynx, nasopharynx, larynx, sinuses, submaxillary lymph

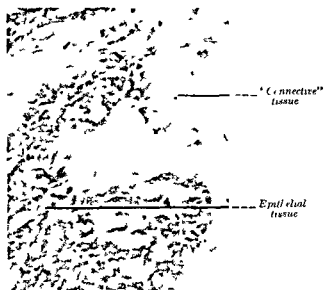


FIG. 326.—Mixed tumor ( $\times 400$ ). The epithelial cells are surrounded by a mucoid connective tissue. On the right cartilage is present.

nodes, lips, cheek, neck, lacrimal gland, etc. Sonnenschein,<sup>2</sup> in 1930, found about 50 cases with involvement of the soft palate.

The theory of embryonic displacement is the commonly accepted explanation of the origin of these tumors. Twing does not classify them as true teratomas. The mucous tissue and cartilage may develop by metaplasia, from gland epithelium.

They may occur at any age and in either sex, however the majority occur during the fourth decade.

<sup>1</sup> Ann. Otol. Rhinol. and Laryngol. 46: 212 (March) 1937.

<sup>2</sup> Arch. Otolaryngol. 11: 137 (February) 1930.

The myxomatous form has a tendency to recur after removal assuming malignant characteristics

**Pathology** —The neoplasm as a rule is a smooth hard circumscribed and somewhat movable growth with a non ulcerated surface Tumors with much cartilage are of firmer consistency than those containing mucus which may be semisolid or cystic The tumors may fill the pharynx or nasopharynx pushing the palate forward Tumors of the hard palate may extend into the nose or antrum and produce a hard swelling of the cheek (New)

Microscopically the tumor is thought to consist only of ectodermal elements (Boyd) and in one place or another the following elements usually are found (1) masses of epithelial cells often showing glandular arrangement (2) mucoid connective tissue with evident production of mucin (3) "cartilage, and (4) lymphoid tissue The tumor epithelial cells produce mucin and this constitutes the mucinous connective tissue A cartilage like appearance is produced by the more homogeneous myxomatous connective tissue From this point of view the mixed tumor should more correctly be called a benign epithelial growth with mucoid degeneration which results in the production of a hyaline material like cartilage

The question as to whether these tumors are benign or malignant has not been fully decided If incompletely removed the myxomatous type tends to recur

McFarland<sup>1</sup> in reviewing 300 mixed tumors of the salivary glands found 69 recurred There were 13 fatal cases Recurrence occurred from 0 to 47 years after operation

**Symptoms** —The symptoms are those of a slow growing mass usually painless or with slight discomfort in the early phase As the mass becomes larger obstructive symptoms such as difficulty in swallowing tinnitus or difficulty in breathing may supervene Ulceration is absent except in a very late stage when an extension through the capsule may occur

**Treatment** —The treatment is complete removal with the capsule According to McFarland the removal should be postponed until the tumor is the size of a lemon as the smaller tumors recur more rapidly New advocates removal as soon as the diagnosis is made if complete removal is possible Many of these tumors may be removed through the mouth Incision is made over the growth and the growth shelled out with the finger or by blunt dissection If the tumor is friable and fixed, it may have to be removed by morcellation If the tumor is large and fixed an external operation through the neck may be necessary usually after ligation of the external carotid artery

The cautery or diathermy should be used on any suspicious areas that are left If the tumor is so extensive that removal is impossible it may be destroyed by the cautery or diathermy with the supplementary use of radium or roentgen ray

### CYLINDROMA

Cylindroma is a special type of mixed tumor somewhat similar to basal cell carcinoma. It is most frequently found in the salivary and lacrimal glands but has been reported as occurring in the nasal sinuses, pharynx, trachea and lungs.

The slow growing, locally malignant tumor is encapsulated, sessile and covered by an intact mucous membrane. There is a marked tendency to recurrence.

Treatment is diathermic excision followed by radiotherapy.

### MELANOMA

Malignant melanoma of the nose and throat is rare. Ringertz<sup>1</sup> in 1938 collected 56 cases including his own. Kaplan<sup>2</sup> reports 2 cases.

Melanoma is a pigmented tumor arising from specific mesoblastic cells, the chromatophore or from tactile or nerve cells of the epidermis (Ewing).

Malignant melanoma of the nose may involve the septum or lateral wall including the turbinates. The lesions appear as a black soft mass which bleed easily on touch.

Symptoms are epistaxis and obstruction to breathing. Metastasis occurs in about 50 per cent of the cases.

<sup>1</sup> Acta Otolaryngol. Suppl. 27 pp. 1-40, 1938.

<sup>2</sup> Arch. Otolaryngol. 35: 85 (January) 1942.



## CHAPTER XXXIV

### MALIGNANT NEOPLASMS OF THE LARYNX

#### CARCINOMA OF THE LARYNX

**Etiology**—Carcinoma of the larynx occurs in about 1.8 per cent of all malignancies of the body. Carcinoma forms about 98 per cent of the malignant tumors of the larynx. About 96 per cent arise from the stratified surface mucous membrane of the larynx and consist chiefly of the squamous cells which form typical squamous-cell epitheliomas or acanthomas. About 2 per cent of epitheliomas of the larynx are basal-cell tumors. About 1 per cent of carcinomas of the larynx are papillary carcinomas. About 1 per cent arise from the mucous glands in the larynx and are known as adenocarcinomas.

Practically all carcinomas of the larynx are of the adult squamous-cell variety. These squamous-cell growths are characterized by slow growth, dilatory extension and usually are irradiation resistant.

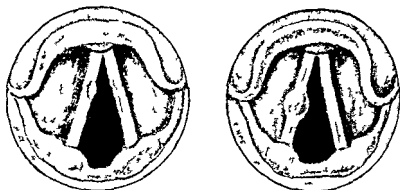


FIG. 3.—Two types of early carcinoma of the vocal cord.

The exciting cause of malignant neoplasms of the larynx is not known. Chronic inflammation of the larynx seems to be a factor, as the statistics show that families having a history of malignant growths are more often attacked in the larynx when subject to chronic inflammation. Papillomas, keratosis and leukoplakia are noted as predisposing to a cancerous condition as well.

As other possible predisposing causes, vocal abuse, the irritant effects of tobacco and ingesting hot drinks and food have been mentioned. Tucker found excessive use of the voice a factor in 41 per cent of his cases and the excessive use of tobacco in 12.5 per cent.

**Heredity**—Heredity is a factor in many cases of cancer of the larynx as in cancer in other locations.

**Age**—The age at which malignant growths of the larynx appear varies somewhat with the variety of the cancer. Sarcoma often occurs

in the very young. It is however more frequent in young adult life. Carcinoma occurs chiefly between the ages of forty and sixty but may be found in rare instances in very young adults.

**Sex.**—Intrinsic cancer of the larynx is about ten times more frequent in men than in women. Extrinsic cancer (excluding the postcricoid form) is only occasionally seen in women.

Mackenty<sup>1</sup> as well as other observers have found laryngeal cancer in the young more common in women than in men, the reverse of the ratio in later life. When present cancer of the larynx in the young seems to be highly malignant.

**Civilization.**—The conditions in life seem to influence the occurrence of malignant growths of the larynx, the civilized nations being more often afflicted than the uncivilized.

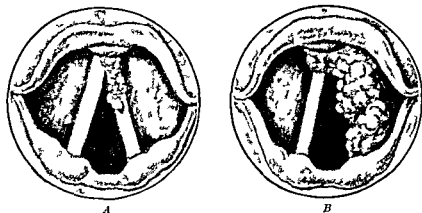


FIG. 38.—A. Carcinoma of anterior half of the left vocal cord. A beginning mass on of the anterior commissure is present. B. Extensive intrinsic carcinoma of the larynx.

**Pathology.**—Cancer of the larynx is usually classified according to the situation as intrinsic, extrinsic, subglottic and mixed.

Intrinsic cancer has its origin in the vocal cords, the ventricular bands and the ventricular pouches. Extrinsic cancer of the larynx arises from the arytenoid cartilages, the epiglottis and other parts contiguous to the larynx.

In intrinsic cancer the growth develops slowly and extends with extreme reluctance by metastasis to the lymph nodes behind the sternocleidomastoid and to the neighboring tissues surrounding the larynx. The intrinsic form is the most common, occurring in about 70 per cent of the cases. It occurs most often in the anterior two-thirds of the true vocal cord, the adjacent portions of the ventricles of the larynx and the anterior commissure. It never appears on both cords simultaneously.

Chordal cancer may occur in the form of papillomas, shallow ulcers or localized thickenings of the cord. There is a tendency to shade off into the surrounding tissue without an inflammatory area around the growth. Immobility of the cord is a fairly late sign. The growth usually

<sup>1</sup> Arch. Otolaryngol. 20:297 (September) 1934.

infiltrates anteriorly and extends to the posterior third only when extensive. If it spreads to the anterior commissure it usually extends to the subglottic region. Intrinsic cancer seldom arises in the posterior commissure or in the interarytenoid tissues. It will develop occasionally in the ventricle usually on the under side of the false cords. Cancers originating in the ventricular bands or in the ventricles form from 5 to 10 per cent of the intrinsic cancers. In this form early invasion of the lymph nodes may occur.

Chordal cancers as a rule do not involve the lymphatics in the early stage as the cords have a very poor lymphatic supply.

The intrinsic carcinoma is a rule is a squamous-celled epithelioma with an occasional basal celled epithelioma. Adenocarcinomas may be encountered in the ventricles.

The extrinsic cancers usually arise from the epiglottis, the aryepiglottic folds, the arytenoids, the pyriform sinuses, the pharyngeal surface of the

cricoid cartilage or as an extension of an intrinsic growth. They are almost always squamous celled epitheliomas with an occasional basal-celled carcinoma. The lymph nodes are involved early. The course of the disease is rapid and surgery seldom cures it.

A subglottic growth is located on the under surface and inner border of the vocal cords or in the subglottic region.

Anteriorly it may extend through the cricothyroid membrane or posteriorly to the arytenoids and the pharyngeal mucosa or superiorly into the larynx and the base of the epiglottis. They may become extensive before



FIG. 309.—Extensive intrinsic carcinoma of the larynx with edema of the epiglottis and left arytenoid.

recognition. As a rule they are squamous-celled epitheliomas.

The mixed form is usually in advanced or late stage of one of the preceding types.

In the beginning subsurface cancers have none of the foregoing characteristics and the advance may be considerable before they appear. Infiltration into the muscles causing diminution in mobility of the affected cord may antedate any diagnostic surface change. Fixation of the cord indicates an advanced lesion with infiltration.

**The Lymphatic Drainage of the Larynx**—The lymphatics of the larynx are of clinical importance in malignant neoplasms and infectious diseases of the larynx.

The lymphatic trunks which take their source from the larynx are derived from a network of radicles which extend throughout the larynx beneath the mucous membrane. This network is divided by a horizontal plane at the level of the vocal cords into a supraglottic and an infraglottic portion. The supraglottic portion includes the lymphatics of the epiglottis, arytenoids, ventricular bands, ventricles and vocal

cords The network of vessels is continuous throughout these areas Over the upper portion and posterior surface of the epiglottis the network is fine and the meshes are far apart In front and lower down especially at the sides the meshwork is denser and the strands thicker Over the arytenoids ventricular bands and throughout the ventricular pouches the lymph channels are thick and closely woven In the vocal cords however the network is very fine and more sparse than in any other part of the larynx The infraglottic network is finer than that above the vocal cords but by no means as fine as that of the cords themselves The lymph from these radicles is collected into trunks which leave the laryngeal cavity at certain definite places

In the upper part of the larynx the only place of egress is through the thyrohyoid membrane The lymph vessels of the upper network assemble in the vicinity of the aryepiglottic folds into several trunks three to six in number which leave the larynx through the above-mentioned membrane near the superior thyroid artery a corresponding group being on either side of the larynx

These trunks course outward and backward more or less in relation to the superior thyroid artery to the carotid region and terminate in nodes which lie along the surface of the internal jugular vein at the level of the bifurcation of the carotid The upper trunk of this group often runs backward after emerging from the thyrohyoid membrane along the hyoid bone to the tip of the lesser cornu and thence outward to a node lying on the inferior aspect of the posterior belly of the digastric muscle The lower trunks of this group may run by a lower course outward and downward into nodes

in the chain lying on the surface of the internal jugular vein below the lower border of the lateral lobe of the thyroid gland (Fig 330)

The collecting trunks of the infraglottic network are divided into an anterior and a posterior division The anterior division consists of three or four small trunks which pierce the cricothyroid membrane in the median line and terminate in small nodes which lie in the median line at uncertain locations The uppermost of these is fairly constant and lies in the V shaped space of the cricothyroid membrane formed by the inner border of the thyroid isthmus and a third on the anterior surface of the trachea These two are denominated respectively the pre

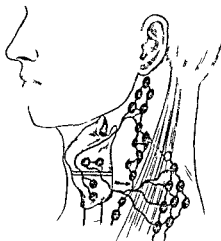


FIG 330—Schema of the lymphatic flow from the supraglottic and the infraglottic regions of the larynx The nodes of the supraglottic region flow into the posterior chain while the infraglottic nodes flow into the anterior cervical chain This is of diagnostic significance in determining if a cancer is supraglottic or infraglottic

thyroid and the pretracheal nodes. They may receive trunks from the anterior infraglottic group. Different trunks from these nodes run to the before mentioned chain lying on the anterior external surface of the internal jugular vein.

In the posterior divisions are three to five infraglottic collecting trunks which penetrate the cricotracheal membrane at or near the line of junction of the cartilaginous and membranous portions of the trachea and run into a chain of lymph nodes two to five in number which lie along the course of the recurrent laryngeal nerve known as the recurrent chain. From these nodes run vessels communicating with the lower most nodes of the internal jugular chain and a few to the supraclavicular group.

The lymphatic drainage from all parts of the larynx thus eventually leads into the chain lying under the sternomastoid muscle along the surface of the internal jugular vein or into the supraclavicular group. The prelaryngeal prethyroid and pretracheal nodes are mere interceptors of the current on its way to the deeper nodes.

The spread of infection or of malignant neoplasms from either the supracordal (glottic) or infracordal region is to the deep lymphatic nodes along the internal jugular vein beneath the sternomastoid muscle or in other words to the same lymphatic system into which the tonsils drain. In infectious and advanced malignant processes of the larynx the deep cervical node along the internal jugular vein and beneath the sternomastoid muscle are enlarged.

**Symptoms** — The earliest symptom may be only a hoarseness intermittent or constant later the chief symptoms are Continued hoarseness without other known cause local discomfort in the larynx slight impairment of the vocal cord on the affected side.

The late symptoms and signs are aphonia dysphagia dyspnea pain in the ear fetor hemorrhage salivation lymph node involvement emaciation cachexia and an immovable vocal cord.

As edema develops and the growth encroaches upon the lumen of the glottis dyspnea of greater or less intensity may embarrass the patient.

Cough increasing with the progress of the disease is usually present. The expectoration is at first similar to that in chronic laryngitis and later is admixed with purulent secretion and with blood in the ulcerative stage.

Dysphagia or difficult deglutition is a late symptom in the intrinsic variety of the disease. If however the primary cancer is in the pharynx or the esophagus it may appear at a much earlier period.

The enlargement of the lymphatic nodes of the neck is a late symptom only occurring after metastasis of the tumor has taken place. Epithelioma is often attended with a very tardy enlargement of the lymph nodes.

**Diagnosis** — The diagnosis of carcinoma of the larynx is made from the symptoms and appearance of the larynx as viewed by indirect or direct laryngoscopy and from a section of the tissue taken by biopsy. It seems to be the consensus of opinion that a biopsy is harmless if the

growth is removed immediately afterward. However, New and Fletcher<sup>1</sup> as the result of microscopic examination of 100 larynges removed for carcinoma of the larynx found that when biopsy had been done some time before actual removal of the larynx there was a slightly greater incidence of microscopic extension.

The biopsy specimen should consist of a portion of the growth including an edge of the normal and a portion of the base of the lesion. If the report is negative in spite of the suggestive clinical appearances another specimen should be secured.

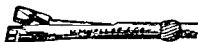


FIG. 331.—Specimen forceps to fit universal handle. The side jaw will bite into a flat lateral wall. The cross forms the bottom of a basket to hold the tissue removed.

The microscopic diagnosis is not always positive for cancer as the typical cancerous growth may be deeply seated beneath the mucous membrane. Negative results should not necessarily be taken as final.

**Differential Diagnosis.** Cancer of the larynx should be differentiated from Chronic laryngitis, syphilitic laryngitis, tuberculous laryngitis, perichondritis, benign neoplasms of the larynx, prolapse or hernia of the ventricles, and unilateral laryngeal paralysis. In rare instances scleroma, blastomycosis, hyperkeritosis, and pachydermia laryngis should be excluded. All cases of hoarseness of doubtful etiology should have a Kahn or similar test to rule out syphilis and a chest roentgenogram. The latter will eliminate primary tuberculous lesions if present and will reveal any lung metastasis from a laryngeal neoplasm. In doubtful cases a biopsy should be done.

In chronic laryngitis the hoarseness, while present in both chronic laryngitis and carcinoma, is not as persistent as in carcinoma. The voice is husky upon arising but becomes clearer during the day. In the hyperplastic variety there are discrete enlargements of the mucosa but they do not have the distinct nodular surface which is present in carcinoma. In chronic laryngitis the vocal cords are freely movable in both abduction and adduction, whereas in late carcinoma one of the cords is immovable. Ulceration is seldom present in chronic laryngitis.

In syphilitic laryngitis the hoarseness is low pitched and brassy or *raucous in character*. In carcinoma of the larynx it is higher pitched and softer in character; indeed it may become aphonic in the later stages. The cords are freely movable in syphilitic laryngitis as a rule. Pain and discomfort are usually absent in syphilis. The history of the case and diagnostic tests usually clear the diagnosis.

Tuberculous laryngitis is characterized by hoarseness and pain, and when perichondritis is present by fixation of one or both vocal cords. In carcinoma one cord only is involved except in the very late stage. A pale edema around the arytenoids is frequently seen in tuberculosis. This is not found in cancer or syphilis. The history and the examina-

<sup>1</sup> Jour. Am. Med. Assn. 99:1754 (November 19) 1932.

tion of the lungs and sputum render the diagnosis so clear that malignancy is practically excluded.

Benign neoplasms of the vocal cords (the most frequent site of intrinsic malignant neoplasm) are characterized by hoarseness though pain and paralysis of the laryngeal muscles are usually absent.

The cells of the malignant epithelial tumors are characterized by a diversity of outline—oval, round, etc. The nucleus is always large and may be single or multiple, staining well with basic dyes. Blood vessels are distinct. A papilloma is an outgrowth of epithelium, whereas a carcinoma is an ingrowth of epithelium.

**Prognosis**—The expectancy of life in the average case of untreated cancer is approximately two years. The prognosis depends in a large measure upon the early recognition and surgical removal of the diseased tissue.

Statistics have shown a remarkable increase in the apparent cures obtained in recent years both by laryngofissure and total laryngectomy.

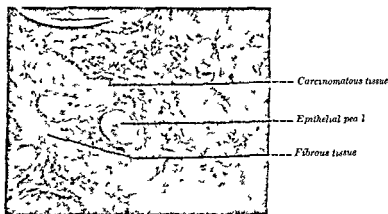


FIG. 33°—Epidermoid carcinoma, Grade I (X 100)

In those cases diagnosed and operated on in the early stage when the lesion is confined to the anterior third of the vocal cord, the prognosis is good as cures are reported in about 80 per cent of selected cases when operated by laryngofissure. In those cases operated on in the late stages the prognosis is bad. Total laryngectomy is attended with greater shock and a higher mortality than the more limited operations. It should be remembered, however, that this method of operating is usually adopted in the more advanced and hopeless cases.

**Cell Types**—The histologic grading of tumors according to their degrees of malignancy by Broder's method seems to have a definite relationship between the cell type of tumor and the prognosis. The points taken into consideration are anaplasia, hyperchromatism, and the number of mitotic figures. The formation of epithelial pearls or presence of much fibrous tissue is evidence of less malignancy. At least we can differentiate between two extremes, that is the low grade malignant squamous-cell carcinoma and the high grade anaplastic type with

numerous mitotic figures. The great majority of cases fall in the large intermediate group.

In many cases of carcinoma of the anaplastic type extensive metastasis may occur early, whereas in those of low grade malignancy years may elapse without any evidence of involvement of the regional lymph nodes.

The cell type of a tumor should be considered in conjunction with the age of the patient, the location and extent of the lesion and the duration of the disease in determining the prognosis and the form of treatment to be carried out.

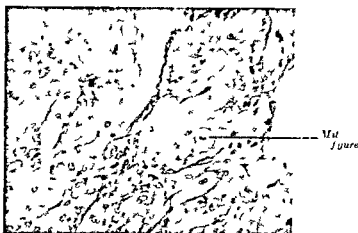


FIG. 333. Epidermoid carcinoma, Grade IV ( $\times 400$ ).

## SARCOMA OF THE LARYNX

**Etiology**—Sarcoma of the larynx is very rare as many neoplasms of the larynx formerly thought to be sarcoma are considered of epithelial origin at the present time. Figg<sup>1</sup> reports out of 717 cases of malignancy of the larynx encountered at the Mayo Clinic only 4 cases of sarcoma were seen.

When present sarcoma of the larynx has been found much more frequently among males than among females. The majority of the cases are over forty years of age.

The etiology of sarcoma of the larynx seems to be the same as that of carcinoma and will not be reviewed further.

**Pathology**—Sarcoma may originate in any portion of the larynx but is most often primary in the vocal cords, especially the anterior half. The subglottic region is stated by some writers to be the most common site.

The neoplasms are pedunculated as a rule. They may be lobulated. Ulceration does not develop ordinarily. Sarcomas of the larynx as a rule have greater tendency to be localized than carcinomas and have less tendency to infiltrate or to develop metastasis.

The spindle celled sarcoma is the type most frequently found. Fibro-

<sup>1</sup>Arch. Otolaryngol. 18:21 (July) 1933.



angiosarcoma chondrosarcoma fibrosarcoma and lymphosarcoma are infrequent

Under the microscope a sarcoma usually appears as a rapidly growing mass of cells. These cells show all forms and shapes but as a rule are uniform mononuclear and have no orderly arrangement at all. There is very little fibrous stroma and the blood vessels possess very indistinct walls so that clinically hemorrhage is frequent



FIG. 334—Pedunculated sarcoma of the larynx growing from the left ventricular band



FIG. 335—View of the inferior surface of the pedunculated sarcoma of the larynx. The peduncle was tubular and composed of mesothelium



— Fibroblasts

FIG. 336—Fibrosarcoma ( $\times 400$ ). The predominant cells in fibrosarcoma are the fibroblasts. The cells are fusiform and may be large or small. The small cells are more malignant. Mitotic figures are visible.

**Symptoms** —The symptoms of sarcoma of the larynx are, as a rule, insidious in onset. Hoarseness develops early, usually in cases in which the neoplasm is situated in the region of the glottis. Dyspnea and respiratory difficulty occur as the tumor enlarges. Dysphagia may be a prominent symptom if the growth involves the epiglottis or a portion of the superior laryngeal border. However, pain or discomfort in the throat is, as a rule, absent or not a prominent feature.

**Diagnosis** —The tumor should be differentiated from a benign growth. The absence of cervical lymph node enlargement in the presence of a rapidly growing and usually pedunculated tumor may lead one to believe the tumor is non malignant.

A positive diagnosis is possible only from a biopsy.

**Prognosis** —The prognosis in sarcoma of the larynx in general is better than in carcinoma since a higher percentage of sarcomas are pedunculated and there is less tendency to infiltration and metastasis.

**Treatment** —The treatment of sarcoma of the larynx depends on the situation, method of attachment, type of growth and its activity. It may be removed by thyrotomy followed by surgical diathermy and irradiation. In more extensive involvements a two-stage operation is advisable (Fig.). Preliminary tracheotomy is performed followed by a later thyrotomy. Extensive tumors are removed by laryngectomy.

## CHAPTER XXXV

### SURGICAL TREATMENT OF CANCER OF THE LARYNX

**Indications** — The surgical removal of a malignant growth of the larynx is indicated if there is a reasonable chance of removing the tumor (and metastasis if present) in its entirety.

**Types of Operations** — Various surgical procedures have been proposed for the different types, locations and extent of malignant growths of the larynx.

As a rule reliance should be placed upon a laryngofissure or a total laryngectomy in most instances.

Laryngofissure is indicated in early intrinsic cancer limited to a small growth in the middle or anterior third of the cord or to growths in the anterior commissure. If the growth has reached the posterior commissure even laryngectomy yields poor results.

Laryngofissure is inadequate if metastasis has occurred or if the growth has extended to the thyroid, arytenoid or cricoid cartilages as sufficient removal of the surrounding normal tissue is not possible by this method. Also in cancer of the epiglottis, aryepiglottic fold or in lesions originating in the infraglottic portion of the larynx laryngofissure is contraindicated.

A growth in the anterior commissure of low grade malignancy should be taken care of by Jackson's technic in which a thyrotomy is performed with removal of the diseased tissue.

Laryngectomy should be done in selected cases if a recurrence has occurred after a laryngofissure in subglottic growths, in extensions to or in involvement of the ventricular bands or arytenoids.

A one stage laryngectomy (Mackenty, Babcock, etc.) may be done unless a preliminary tracheotomy is necessary for the relief of dyspnea. Partial laryngectomy (thyrochondrotomy of Sir St. Clair Thomson) with wide excision of the affected part may be performed on patients who refuse laryngectomy or where the general condition does not permit a total laryngectomy. New<sup>1</sup> advocates a two-stage thyrotomy in cases of carcinoma of the larynx which are considered bad risks.

Carcinoma of the aryepiglottic folds, arytenoids, postcricoid area and pyriform sinuses contraindicate a laryngectomy as a rule as intrinsic growths that have extended to these areas render the growth inoperable in most instances. Other contraindications are cardiovascular disease, tuberculosis, nephritis and diabetes. Radiation rather than laryngectomy should be used in these cases as a rule.

The endoscopic removal of cancer of the larynx is justifiable when the extreme tip of the epiglottis is involved in an early, small malignant growth. The entire epiglottis is amputated. A low-grade cancer definitely limited to the epiglottis can be removed by performing a

<sup>1</sup> Arch. Otolaryngol., 9: 533 (May) 1929.

preliminary tracheotomy. Then by means of a Lynch suspension apparatus the growth is removed by diathermy under direct vision. If the growth extends laterally to the aryepiglottic fold and the arytenoid region, a subhyoid pharyngotomy is a better procedure (New<sup>1</sup>). A pharyngotomy may be performed through the thyrohyoid membrane after division and retraction of the hyoid bone or as a lateral pharyngotomy. In some cases a window is removed from the thyroid cartilage on the uninvolved side permitting the growth on the opposite side of the larynx to be destroyed with surgical diathermy through this opening. A cancerous invasion of the cervical lymph nodes usually contraindicates surgery.

### LARYNGOFISSURE THYROTOMY

**Jackson's Operation.** **Anesthesia.** General anesthesia as a rule is unnecessary for laryngofissure. If given the intratracheal insufflation of ether is best. Jackson\* uses local infiltration with procaine solution until the cartilage is exposed and the external perichondrium incised. Then through the incision made in the cricothyroid membrane gauze sponges saturated with 10 per cent cocaine solution are inserted. If preferred procaine solution can be injected between the mucosal surface and the perichondrium. A sedative given one and a half hours before the operation permits the effect to have worn off by the time the operation is finished so that the cough reflex will have returned.

**Incision.**—A central skin incision is made from the thyroid notch to just below the cricoid. Flaps are not dissected or the larynx is not skeletonized. The incision is carried down to the cartilage. Hemostasis is secured. A transverse incision is made in the cricothyroid membrane and two small sponges saturated with 10 per cent cocaine solution are passed through this opening by means of forceps and kept in contact with the interior mucosa about two minutes.

The external perichondrium is incised in the mid line. A hand or rotary saw is used to cut through the thyroid cartilage in the mid line so as not to go through the internal perichondrium. In using the saw care should be taken to not let the saw heat. If the growth is in the middle of the cord the shears may be used to cut the cartilage. The lower blade is inserted through the incision of the cricothyroid membrane taking care that the cutting is not done toward the involved side.

**Elevation of the Internal Perichondrium.**—The perichondrium is elevated slightly using the point of a special Jackson tenaculum to hold the edge of the cartilage on the side uninvolved or least involved. Dissection is carried back beyond the extent of the growth. The thyroid wing on this side is now held back with a retractor using care not to let the retractor slip or some of the external perichondrium may be removed causing a necrosis of that portion of the cartilage. At times it is necessary to extend the subperichondrial dissection to the arytenoid cartilage.

<sup>1</sup> Arch. Otolaryngol. 24: 485 (October) 1936.

\* Am. Jour. Surg. 30: 3 (October) 1935.

**Excision of the Growth**—The growth is excised with a wide area of normal tissue. A portion of the ventricular bands may be included even though only the cords are involved. An ample area of the subglottic normal tissue should be included.

**Closure of the Wound**—The soft tissues are closed. The skin is closed with clamps, silkworm gut, silver wire or non-oxidizable steel wire.

A strip of iodoform gauze secured with a suture may be placed in the lower angle of the wound. A second strip of gauze may be placed in the middle of the incision down to the cartilage. This later drain is removed in twenty-four hours. The lower angle of the wound is kept open until the incision in the cricothyroid membrane closes.

A light dressing of two or three layers of gauze is applied.

Tracheotomy may be required if necessary to pack the larynx on account of hemorrhages or for other reactions in the larynx.

Sir St. Clair Thomson dissects the thyroid wing on the affected side free of perichondrium internally and externally and excises the freed portion of cartilage. This gives good exposure of the inside of the larynx.

**Two stage Thyrotomy**—The two-stage thyrotomy is advocated by New<sup>1</sup> in carcinomas of the larynx which are considered bad risks.

**First Stage**—One-half per cent procaine is infiltrated into the median line of the neck from above the hyoid bone to just below the cricoid cartilage and down about the hyoid bone and laterally to the thyroid cartilage. The incision is then made in the median line and the hyoid bone divided. The ends are retracted laterally by means of sharp retractors. The muscles are separated from the anterior portion of the larynx down to the cricoid cartilage. The wound is then closed with dermal sutures.

Four days later a small opening is made into the cricothyroid membrane or by removing a disk from the cricoid cartilage into which a small tracheal tube is inserted if necessary to maintain the opening.

**Second Stage**—About four or five days later under block paravertebral anesthesia the wound is opened and the divided ends of the hyoid bone retracted. The upper end of the thyroid cartilage is packed off and divided by means of a saw. The cricoid cartilage is not disturbed.

The thyrohyoid membrane is partially divided in the median line for better exposure. The laryngeal growth is removed by diathermic dissection.

The thyrohyoid membrane is closed and the muscles sutured over the thyroid cartilage. A split rubber tube is placed at the lower end of the wound and left in place for forty-eight hours.

## LARYNGECTOMY

**Anesthesia**—Local anesthesia is usually used by infiltrating the skin and tissues of the neck with a 1 per cent solution of procaine. A basal anesthesia of avertin per rectum supplemented by the intravenous injection of sodium penthotal is used in addition by most laryngologists.

<sup>1</sup> Arch. Otolaryngol. 9:538 (May) 1909

**Incision** — A mid line incision is made from the hyoid bone to the sternal notch

**Division of the Thyroid Isthmus** — The cricoid cartilage and the thyroid isthmus just below the cricoid cartilage are located and the thyroid isthmus is severed clamped and sutured. This exposes the trachea for possible tracheotomy if necessary.

**Skeletonization of the Larynx** The sternohyoid sternothyroid and thyrohyoid muscles are separated in the mid line. The insertions of the sternothyroids and the thyrohyoids into the thyroid cartilage are separated from the thyroid cartilage by subperichondrial dissection to preserve their continuity (Jackson and Norris<sup>1</sup>). The inferior constrictor and other muscles of the pharynx are also severed from the thyroid cartilage. The larynx must be freed down to the esophagus on both sides.

The skeletonization of the larynx may be done subperichondrially as described by Crowe and Broyles<sup>2</sup> if the growth has not reached the thyroid cartilage.

**Resection of the Hyoid Bone** The hyoid bone is usually removed or partially resected which gives a wider space to work and facilitates the repair of the esophagus at a later stage of the operation.

**Severing the Trachea** — The trachea is severed between the first and second rings as a rule and the severed stump sutured to the skin with a temporary suture. An inner cannula is inserted into the tracheal stump to prevent secretions from running into it. Cunnings<sup>3</sup> leaves a tongue of trachea in the shape of an inverted epiglottis to act as a dam to prevent blood from entering the trachea and as a solid wall above the tracheal opening when the wound is closed.

**Separating the Larynx From the Esophagus** The larynx is separated from the esophagus from below. When the superior cornua of the thyroid cartilage are reached they are dissected free or amputated. The superior laryngeal vessels entering the thyrohyoid membrane anterior to the cornua and above the edge of the thyroid cartilage are ligated.

**Opening of the Pharynx** — The pharynx is opened in the posterior mid line. Each side is incised just above the arytenoids and posterior commissure to the valleculæ.

**Removal of the Larynx** — Special clamps (Vasconcelors Barrett<sup>4</sup>) may be applied to the pharynx before it is opened to facilitate the removal of the larynx or Kelly clamps may be placed across the base of the tongue between the tongue and the epiglottis on each side. The remaining attached portion of the larynx is then separated and the larynx removed.

**Closure of the Pharynx** — The upper openings in the pharynx and the upper end of the esophagus are closed in the mid line using inverted

<sup>1</sup> Laryngoscope 55 196 (May) 1945

<sup>2</sup> Ann Otol Rhinol and Laryngol 47 875 (December) 1938

<sup>3</sup> Trans Am Acad Ophthal and Otolaryngol (March-April) 1944

<sup>4</sup> Arch Otolaryngol 40 275 (October) 1944

interrupted sutures of fine silk or fine silver wire. A sterile small size Jutte tube should be passed through the esophagus into the stomach with the proximal end in the pharynx where it is later on brought forward through the nose.

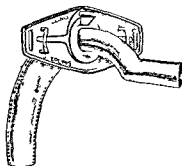


FIG. 337. Orton's laryngectomy tube with elbow extension for artificial larynx.

The supporting tissues are closed by means of a second row of interrupted sutures of fine wire inverting the mucosal edges into the pharynx. The sternohyoid or omohyoid muscles are tacked over the suture lines for reinforcement.

The trachea is now sutured to the skin using heavy linen or wire sutures.

The tracheotomy tube wrapped with gauze covered with petroleum jelly or bismuth paste is then inserted.

**Drainage and Dressing**—Small drainage tubes are placed in the lower incision or the lower 2 inches of the wound may be left open for drainage.

Split pads of gauze are placed in front of the neck and around the tracheal cannula.

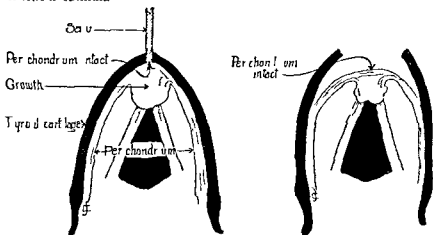


FIG. 338. Jackson's method of subperichondrial dissection in anterior commissure growths. Procedure in cases in which the cancerous growth is in the anterior commissure. The shears are not used for fear of cutting the growth. The thyroid cartilage is sawed through without injury to the inner perichondrium which is then dissected backward safely beyond the posterior limits of the growth (Tucker Arch. Otolaryngol.).

**MacKenty Technic** — The I shaped incision of MacKenty or the straight mid line incision may be used. The I shaped incision as advocated by MacKenty and others is especially useful. The difficulty is to secure a primary union at the point where the two lines of the I cross otherwise a hypopharyngeal fistula may result.

Intratracheal inhalation anesthesia may be used or a combination local and general anesthesia as used by MacKenty may be preferred.

The mid line incision begins just below the hyoid bone and extends to the second ring of the trachea. When the larynx has been skeletonized the gauze is removed from the oropharynx and all secretion sucked out. The intratracheal inhalation tube is removed.

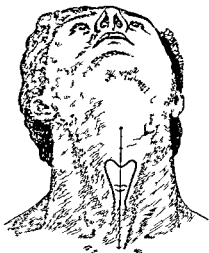


FIG 339 — The line of incision for the complete or partial removal of the larynx.

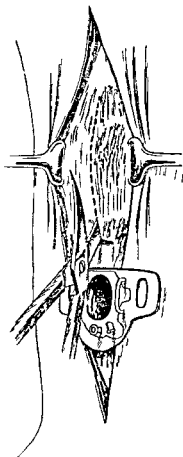


FIG 340 — Laryngofissure Tracheotomy has been performed a cross-puncture at the lower border of the thyroid made and the scissors blade introduced through it preparatory to making the incision through the anterior commissure of the thyroid cartilage.

The trachea is cut through to the anterior esophageal wall and partially separated posteriorly. The MacKenty conical rubber tube is placed in the tracheal opening and the anesthetic is continued through this. Buckley suggests the immediate tacking of the trachea to the skin at the time of the tracheal incision which prevents leakage of blood into the chest and makes it possible to proceed without the formerly used rubber hose dam.



A strip of iodoform gauze is packed through the larynx into the hypopharynx.

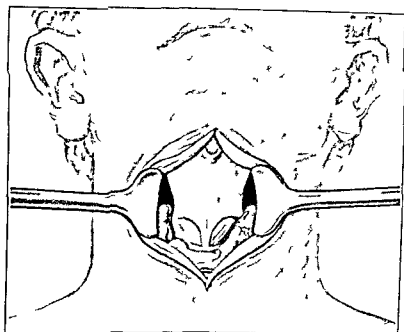


FIG. 341.—Total laryngectomy. The thyroid cartilage exposed.



FIG. 342.—Extrinsic carcinoma of the larynx. Posterior view.

The larynx is separated from the esophagus from below upward to a point behind the arytenoids. The thyrohyoid membrane is divided.

If the growth is intrinsic the larynx is removed by cutting close to the superior border of the thyroid cartilage. After the larynx is removed the esophageal opening and that in the hypopharynx are closed.

A feeding tube is then introduced through the nose and passed down into the esophagus about 8 inches where the tube is fixed in position.

The tracheal tube is now removed and the trachea is attached to the skin by means of interrupted sutures.

Rubber catheter drain tubes are inserted. The mid line incision is then closed with parallel mattress sutures of silk worm gut. A large tracheal cannula is wound with gauze impregnated with bismuth paste and inserted into the trachea to prevent wound secretions from entering. The cannula is continued until the wound is healed.

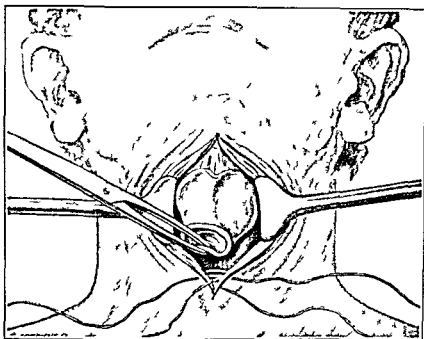


FIG 313 Total laryngectomy The thyroid cartilage is severed from the trachea

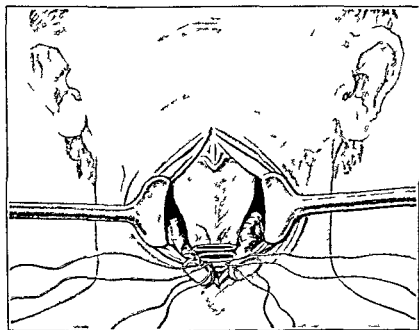


FIG 344 —Total laryngectomy The isthmus of the thyroid gland is severed and retracted

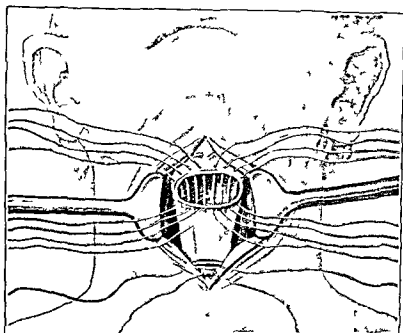


FIG. 345.—Total laryngectomy. The larynx has been removed and sutures placed in the pharyngeal opening.

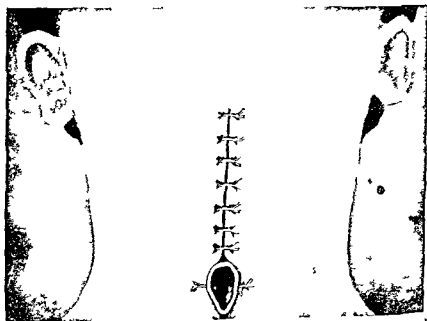


FIG. 346.—The incisions after complete laryngectomy. The end of the trachea is sutured to the skin.

**Partial Laryngectomy**—This operation is often spoken of in the literature as synonymous with laryngofissure which is but the preliminary step in partial laryngectomy. Partial laryngectomy is a more extensive operation than simple laryngofissure. In laryngofissure only the soft parts and the growth are removed whereas in partial laryngectomy a portion of the cartilaginous framework is removed with the growth (Fig 347)

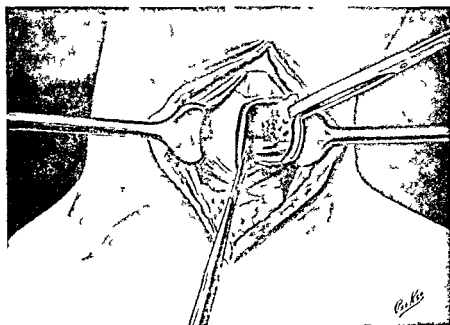


FIG 347 —Partial laryngectomy for intrinsic carcinoma of one side of the larynx

Norman Patterson<sup>1</sup> finds partial or anterior laryngectomy rather than complete extirpation is often sufficient for a carcinoma developing from the false cord or ventricle in which the growth has crossed the middle line and has spread to the region of the anterior commissure

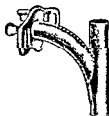


FIG 348 —Jackson's laryngostomy tube



FIG 349 —Koshler's laryngostomy tube

**Technic**—The technic is so little different from that given in laryngofissure that a detailed description is unnecessary. The chief difference consists in the removal of the affected portion of the cartilaginous

<sup>1</sup> Arch Otolaryngol 23 '96 (March) 1936

framework in addition to the procedures practised in laryngofissure in which only soft tissues are removed

Patterson uses a flap incision exposing the whole of the thyroid cartilage on the side chiefly involved with the exception of the posterior border and the cornu. The anterior three fourths of the right thyroid ala and the anterior third of the left ala is removed with punch forceps. The internal perichondrium is separated from the thyroid ala as the removal of the cartilage progresses over a limited area. The tracheotomy tube is inserted and the anesthetic is administered through it. Excision of the growth is now carried out the surface and the edge of the tumor being kept in view.

### ESOPHAGEAL VOICE AND ARTIFICIAL LARYNX

The restoration of understandable speech after a laryngectomy may be accomplished by the development of an esophageal voice or by prescribing an artificial larynx.



FIG. 300 Wolf's artificial larynx

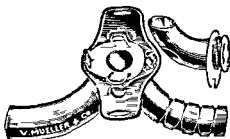


FIG. 301 Von Brun's artificial larynx

The development of an esophageal voice is based on the esophagus acting as an air chamber. The patient creates positive air pressure in his esophagus or possibly stomach by swallowing air. As the patient releases the air against the esophageal glottic folds which act as a substitute glottis phonation is attempted. With practice a good voice may be developed. Some patients develop a voice by throwing the air along the side of the tongue with the base of the tongue closed against the posterior pharyngeal wall.

The artificial larynx consists of a flexible metal reed placed next to a rubber membrane within a metal sound box. The mouthpiece is attached to the top of the sound box and the tracheal stoma to the bottom by a flexible connection. The air is inhaled through a hole in the side of the sound box passing by way of a metal tube in the bottom of the box into the tracheal connection and on into the lungs. The patient places his finger or thumb over the hole in the side of the box when exhaling forcing the air to pass around the vibratory reed and on through the mouthpiece into the mouth where the air is converted into speech.

## IRRADIATION OF MALIGNANT GROWTHS OF THE LARYNX

Irradiation (radium or roentgen ray) treatments of malignant growths of the larynx have been confined to a large extent to advanced lesions that were considered incurable by surgery or as a supplementary treatment to surgery. In recent years reports up to 82 per cent (Cutler<sup>1</sup>) three year survival rate in selected intrinsic growths of the larynx have been made. This three year survival rate is still less than the five year survival rate by laryngofissure in selected intrinsic growths. Blady and

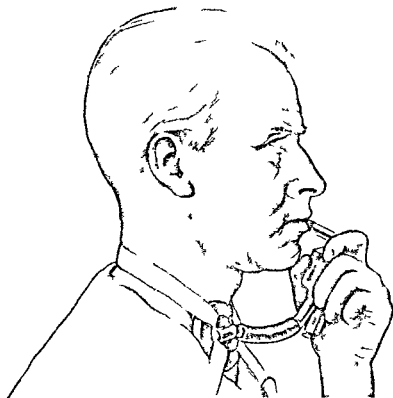


FIG. 3a. —Mackenty Western Electric artificial larynx in use (Mackenty, *Arch Otolaryngol*)

Chamberlain report a five year survival without evidence of recurrence or metastasis in 19 per cent of the intrinsic group and 20 per cent of the extrinsic cases treated by irradiation.

The contraindications to irradiation are (1) the advanced case in which laryngeal obstruction is imminent (2) damaged laryngeal structures from former irradiations (3) if the growth is still present after a

<sup>1</sup> Jour. Am. Med. Assn. 124:967 (April 1) 1944

<sup>2</sup> Am. Jour. Roentgenol. 51:481 (April) 1944

total dosage of 12 000 r has been given (Mc Cormick<sup>1</sup>) and (4) if the response to the treatment is not satisfactory before this time.

The exact indications for irradiation have not been formulated as yet. It may be indicated as a curative or palliative measure in (1) extensive lesions that are not amenable to surgery (2) presence of metastasis in the neck (3) recurrence following surgery (4) as a prophylactic post-operative measure (5) poor operative risk due to advanced age renal or cardiac complications etc (6) if the patient refuses surgery (7) lesions that have reached the posterior end of the vocal cord even though the motility of the cord is not impaired and (8) possibly in high grade radio-sensitive growths of the intrinsic type.

The choice of using radium or roentgen rays depends upon the location and extent of the lesion and upon the individual skill of the operator. High voltage roentgen therapy is chosen in most instances when it can be given by the Coutard type of repeated daily doses (described elsewhere). Howes and Bernstein<sup>2</sup> of the Brooklyn Cancer Institute have calculated a system of roentgen ray dosage that seems acceptable.

If radium is used it is usually in the form of teleoradium. The large amount of radium required and the bulky container makes this method more difficult to use than roentgen therapy. Radium may also be used in the form of seeds or needles for implanting in the tissue. This type is especially useful for implantation through the skin or after a surgical exposure.

<sup>1</sup> *Canad Med Assn J* 50 56 (June) 1914

<sup>2</sup> *Am Jour Roentgenol* 50 6 1913

## CHAPTER XXXVI

### MALIGNANT NEOPLASMS OF THE TRACHEA PHARYNX AND NOSI

#### CARCINOMA OF THE TRACHEA

**Etiology**—Carcinoma of the trachea is rare. It is observed in about 0.004 per cent of all autopsies (Stenn).

D'Aunoy and Zoeller in a search of the literature found a total of 91 instances of primary carcinoma of the trachea. Secondary involvement of the trachea occurs more commonly than the primary type.

Males are more commonly involved than females. The age of greatest frequency is between fifty and sixty years.

**Pathology**—Two forms of primary carcinoma of the trachea are seen: Diffuse infiltration with ulcerations and circumscribed polypoid or fungoid type. The infiltrating form is more common than the polypoid. They are usually of the squamous-cell type with an occasional adenocarcinoma or a cylindroma basal-cell form found. The squamous-cell epitheliomas are usually near the bifurcation and well localized.

Metastases are usually restricted to the tributary lymph nodes, seldom extending to distant organs.

**Symptoms**—The early symptoms of malignant tumors of the trachea are a tickling sensation in the trachea with paroxysms of coughing. Crusts or small clots of blood may be coughed up. Active hemorrhage may occur as a late terminal symptom. Dyspnea may be noted at first on exertion but later may become constant, especially during inspiration. During the attacks cyanosis may be present. Hoarseness usually develops late in the course of the disease as a result of involvement of the recurrent laryngeal nerve.

The prognosis is bad. Death is usually due to suffocation or pneumonia.

Asthma, thyroid disease, emphysema, bronchiectasis and pulmonary abscess should be differentiated from malignant tumors of the trachea.

**Treatment**—Lesions in the upper half of the trachea may be treated surgically by tracheofissure and destroying the growth with electrocoagulation. Excision of a segment of the trachea has been advocated. Implanted radium and irradiation over the regional nodes with radium packs or deep roentgen rays after Coutard's technic are indicated in the inoperable cases.

#### MALIGNANT NEOPLASMS OF THE HYPOPHARYNX

Malignant growths of the hypopharynx include all malignant neoplasms originating in the posterior inferior portion of the tongue.



epiglottis valleculæ pyriform sinuses lateral and posterior pharyngeal walls postericoid and interarytenoid regions and the epiglottic folds.

Malignancy of the posterior pharyngeal wall usually is found in women as are the ones occurring in the postericoid region. The latter may occur during relatively early periods of life.

The epipharyngeal group involving the arytenoids aryepiglottic folds epiglottis pyriform sinus and possibly the base of the tongue is limited to men as a rule.

**Pathology**—These neoplasms may be a low grade squamous type or a high grade transitional-cell carcinoma or a lympho-epithelioma. Cervical lymph node metastases are usually present but may occur late in the low grade squamous-cell type.

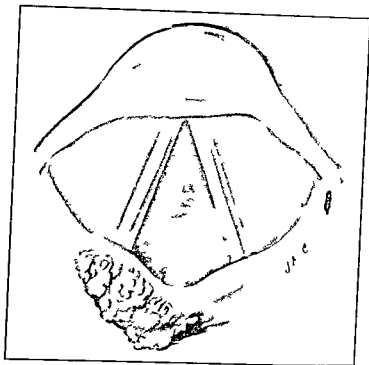


FIG. 353.—Postericoid carcinoma. The nodular growth has extended upward to the pharyngeal surface of the larynx. Swelling of the aryteno-epiglottic folds is usually present. (After Negus.)

Malignant tumors of the epiglottis usually originate from the posterior side near the tip. They often appear as a cauliflower like mass usually sessile but may be pedunculated. As a rule there is little infiltration about the base.

**Symptoms**—The symptoms during the early stages are vague as a rule. Symptoms are produced later than those originating on the vocal cords. Irritation in the throat and a sensation as of a foreign body in that region are early symptoms. Later a sense of fulness and difficulty

in swallowing may develop. The vocal cords may be involved producing hoarseness or dyspnea. At times dysphagia and hemoptysis may occur late in the course of the disease.

If the aryepiglottic fold is involved hoarseness and even dyspnea may be present at an early stage. Growths of the pyriform sinus may give little or no symptoms until later. In these cases marked cervical lymph node enlargement may be present before the symptoms.

A malignant growth of the lateral pharyngeal wall is usually characterized by slow growth.

Examination may reveal a small ulceration or a small neoplasm when the patient is made to phonate strongly. Edema of the arytenoid may be observed.

The diagnosis of tumors in this region is made from the history, the clinical examination and biopsy. Benign tumors should be excluded as well as syphilis and tuberculosis.

Cylindromas or recurrent basal-cell carcinoma of the oral and respiratory mucous membrane have been reported a number of times. There is little agreement with regard to the histogenesis and classification. According to Bredlau<sup>1</sup> this group of tumors is relatively mildly malignant, recurs locally after removal and invades the surrounding structures late if at all.

The cells resemble closely the basal cells of the covering layer of epithelium as such they may be classified as basal-cell carcinomas with the addition of such descriptive terms as solid, cystic or adenoid as suggested by Krompecher.

**Treatment**—The transitional cell and lympho-epitheliomas should be treated with irradiation.

Surgical excision consists of the removal of the growth by trans-thyroid pharyngotomy which permits surgical access to the entire laryngopharynx. A preliminary low tracheotomy is necessary.

A lateral pharyngotomy may be used in the removal of the lateral pharyngeal growths. Operation is contraindicated if an extension to the larynx has occurred.

## MALIGNANT GROWTHS OF THE EPIGLOTTIS

Cancer of the under surface of the epiglottis may be slow growing and very slow to metastasize. If the disease has not penetrated the cartilage and there is no evidence of metastasis removal of the epiglottis should be sufficient to effect a cure.

Four methods of treating cancer of the epiglottis are in common use: (1) Removal of the epiglottis by thermocautery or snare through the mouth by means of suspension laryngoscopy. (2) Excision of the epiglottis by the trans-thyroid approach. (3) Excision of the epiglottis by a lateral pharyngotomy. (4) When the growth extends downward involving the ventricular bands the procedure most commonly employed is total laryngectomy.

If the growths are infiltrating or if there is extension beyond the epiglottis either pharyngotomy or laryngofissure is advisable. Laryngofissure is satisfactory if the growths are situated laterally or posteriorly above the vocal cords but confined within the larynx. Pharyngotomy either subhyoid or tranthyroid usually affords the best view depending on the situation and extent of the lesion. Lesions of the epiglottis extending into the aryepiglottic folds or the pyriform fossæ may be approached through a subhyoid operation, by incision transversely at the base of the tongue and drawing the epiglottis out into the wound. According to Figi<sup>1</sup> when there is limited involvement of one aryepiglottic fold with some extension onto its outer aspect or when there is involvement of the epiglottis and base of the tongue, lateral pharyngotomy, after removal of the thyroid ala and if necessary, of a portion of the hyoid bone is preferable. The lesion is removed by sharp or cautery excision, by electrocoagulation or by a combination of these. Radium element may be implanted as a supplementary measure if it be sufficiently far removed from the laryngeal cartilages to guard against the possibility of perichondritis. Sarcomas of the supraglottic region may respond more satisfactorily to irradiation than to surgical procedures.

If the lesion is extensive a preliminary tracheotomy about two weeks before the operation is advisable. General anesthesia is necessary for suspension.

Following exposure, the lesion may be removed or destroyed by excision, cauterization, electrocoagulation or irradiation.

**Lateral Transthyroid Pharyngotomy**—Trotter's<sup>2</sup> operation for malignant growths of the laryngopharynx is as follows. A preliminary tracheotomy is done. The incision is made along the anterior border of the sterno-cleido-mastoid muscle. The muscles of the larynx are reflected anteriorly and posteriorly. If enlarged lymph nodes of the neck are present from an extension of the growth an additional incision is made from the angle of the lower jaw to the symphysis forming triangular flaps (Orton<sup>3</sup>).

The superficial fascia, submaxillary gland and deep fascia are displaced upward and backward exposing the deep vessels of the neck and the muscle. The vessels extending anteriorly from the great vessels are ligated and cut. The internal jugular vein is then ligated and resected. In some instances it is necessary to ligate the external carotid. The sterno-cleido-mastoid is sutured to the prevertebral fascia covering the great vessels. The muscles are reflected from the hyoid bone and thyroid cartilage. The exposed portions of the hyoid and the greater portion of the thyroid cartilage are removed by means of heavy scissors. The pharynx is opened by an incision to give an approach to either a hypopharyngeal or an epilaryngeal growth.

<sup>1</sup> Arch. Otolaryngol. 20:361 (September) 1934.

<sup>2</sup> Lancet 1:1075 (April 19) 1913; ibid. 1:1147 (April 26) 1913.

<sup>3</sup> Arch. Otolaryngol. 12:320 (September) 1930.

**Removal of the Epiglottis by Thyrotomy**—Tucker gives the technic of removing the epiglottis by a laryngofissure.<sup>1</sup>

A preliminary tracheotomy is done a week or ten days before the thyrotomy. Avertin anesthesia 70 mg per rectum with procaine infiltration, is used. A mid line vertical incision is made from the hyoid bone to a point just above the tracheotomy fistula. The front of the larynx is exposed from the cricoid cartilage to the thyroid notch. The cricothyroid membrane is incised vertically, the thyroid cartilage divided with a turbinotome. The wings of the thyroid cartilage are retracted exposing the interior of the larynx.

A subperichondrial resection of the inner surface of the left wing of the thyroid about 1 cm from the left side of the thyroid notch is done. This excision includes the anterior end of the left ventricular band. The same procedure is carried out on the right side.

The thyrohyoid membrane is divided sufficiently to allow exposure of the base of the epiglottis. The base of the epiglottis is grasped and pulled downward and the mucous membrane of the upper surface of the epiglottis is removed by subperichondrial dissection. This frees the epiglottis except at its attachments on either side. The incisions through the ventricular bands are extended upward and the tissues including the aryepiglottic fold on either side and the epiglottis are removed. The bleeding points are ligated with suture ligatures.

The flap of the mucous membrane which had previously covered the upper surface of the epiglottis is pulled downward covering the denuded area on the front of the hyoid bone where the base of the epiglottis had been attached. A small piece of cartilage is removed from the upper corner of the thyroid wings on either side, the external perichondrium being dissected from the cartilage and left in position. This permits the thyrohyoid membrane and the tissues on either side to be pulled together with a deep silkworm gut suture which is carried around from the skin surface on one side encircling the tissues where the base of the epiglottis has been attached and coming from within outward through the skin on the opposite side. This deep suture closes the opening remaining after the removal of the epiglottis. The laryngofissure wound is closed using interrupted silkworm gut sutures.

**Subhyoid Pharyngotomy**—Subhyoid pharyngotomy for the removal of malignant neoplasms of the larynx is rarely used. There are cases however when it may be elected.

**Indications**—The indications for subhyoid pharyngotomy are few and it is used chiefly in cases of malignant neoplasms complicated by extension to or by origin in the pharynx.

**Technic**—Make a transverse incision through the skin after Kocher's method beginning about  $\frac{1}{2}$  inch below the inferior border of the hyoid bone and extend it from the anterior border of the sterno cleido mastoid muscle to the corresponding point on the opposite side of the neck. The incision should be from  $2\frac{1}{2}$  to 3 inches in length. Then make a per

pendicular incision in the median line beginning above the transverse incision and extending downward to the prominence of the thyroid cartilage.

Divide the superficial fascia in which the anterior jugular vein is found. The jugular vein should be ligated in two places on each side of the neck and severed between the ligatures.

Sever all the muscles including the sternohyoid on either side of the median line and just beneath them the thyrohyoid muscles thus exposing the thyrohyoid membrane to view. With the finger applied to the membrane explore for the epiglottis so as to avoid injuring it in the next step of the operation.

Incise the thyrohyoid membrane thus exposing the diseased area to inspection.

Carefully inspect the deeper field beginning at the anterior surface of the epiglottis for evidences of a malignant growth.

Seize the epiglottis with toothed forceps and gently draw it outward through the wound securing it with either a suture through its tip or with locked forceps. Traction upon the epiglottis opens the wound and exposes the deeper parts to view.

Through the opening all diseased tissue is removed with scissors knives and double cutting forceps some of the surrounding healthy tissue being also included.

The wound is now closed by suturing the thyrohyoid membrane the muscles and the superficial fascia with absorbible catgut and the skin with non-absorbible ligatures.

### MALIGNANT NEOPLASMS OF THE NASOPHARYNX

Malignant tumors of the nasopharynx may be carcinomas sarcomas or endotheliomas. Numerous subdivisions of the first and second groups are mentioned. Under carcinomas are listed squamous scirrhus baso-cellular anaplastic epidermoid or spino-cellular adenocarcinomas transitional cell and lympho-epithelioma. Under sarcomas are listed lymphosarcoma spindle-cell round-cell myxosarcoma fibrosarcoma chondrosarcoma reticulum sarcoma and polyhedral-cell sarcoma (Salinger and Pearlman<sup>1</sup>).

Sarcoma and endothelioma are extremely rare in the nasopharynx. The large majority of the tumors in this location are the anaplastic or transitional-cell type. It is possible many of the tumors diagnosed sarcoma or endothelioma are transitional-cell carcinoma or lympho-epithelioma.

**Etiology**—Cancer of the nasopharynx comprises about 2 per cent of all malignant growths of the head and neck. It occurs much more frequently in the male (about 80 per cent) and occurs at a younger age than cancer in other regions of the upper respiratory or alimentary tracts. The Chinese seem to have an unusual racial susceptibility to malignant growths in this region.

**Pathology**—Lympho-epithelioma and transitional-cell carcinoma show little difference in their development and histologic characteristics depending on the amount of lymphocytic infiltration. Both of the types seem to originate from the transitional epithelium and the lymphoid tissues of the nasopharynx and throat.

The areas in which the transitional-cell epithelium is found most frequently are the nasopharynx, tonsil, base of the tongue, larynx and esophagus. They are found especially in the ducts of the associated mucous glands. Many of these malignant growths have their origin from the epithelial lining of the eustachian tube.

The transitional-cell carcinoma is formed of masses of small cells with a large nucleus occupying almost the entire cell. Variations in the size and staining qualities of the cells are observed. They tend to grow in cords and sheets.

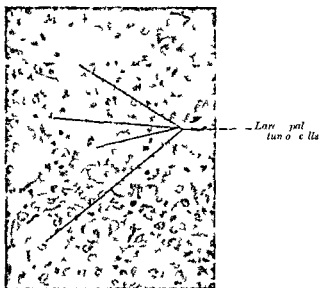


FIG. 354.—Lympho-epithelioma ( $\times 400$ )

The lympho-epithelioma cells are anaplastic with pale protoplasm and vague cell boundaries. A syncytial formation is evident. The nuclei are large, clear and pale with frequent mitoses. The cells seem to grow in sheets or columns with an interlacing supporting stroma. The infiltration of lymphocytes varies in amount.

Ewing in a study of 100 cases of nasopharyngeal malignancies found 37 per cent were of the transitional type, 11 per cent of the lympho-epithelial type and 30 per cent were squamous-cell carcinoma.

The growth in the beginning is small and located beneath the epithelial surface making detection difficult.

Metastasis to the viscera is rare.

Fibrosarcoma usually appears singly. It is rounded and very firm. The rate of growth is usually rapid.

Lymphosarcoma of the nasopharynx arises from the lymphoid tissues. It may extend to the surrounding structures: cervical nodes, the orbit or any lymphoid tissue in the body may become infected.

Reticular-cell sarcoma of the nasopharynx is very rare but is reported as occurring more frequently elsewhere in the body.

**Symptoms**—Malignant growths of the nasopharynx are usually overlooked on account of the absence of symptoms referable to the nose or nasopharynx. It is only in recent years that many cases have been reported due to the recognition of the peculiar syndrome exhibited by these patients.

In 1922 New<sup>1</sup> called attention to the rather characteristic symptom complex with early involvement of the cervical lymph nodes and the symptomless progress of the primary stage.

The earliest symptom as a rule is the painless indurated enlargement of the cervical lymph nodes. The patient may complain of a slight fulness or tinnitus of the ears due to impingement of the tumor upon the mouth of the eustachian tube. The tympanic membrane may have the dull retracted appearance associated with eustachian tube blocking.

Fifth nerve pain located in the temple, forehead, eye, cheek, teeth or vertex is an early symptom.

The next train of symptoms is usually from involvement of other cranial nerves. The cranial nerves most frequently involved are the fifth, sixth, third and fourth and sometimes the seventh. The second and eighth<sup>2</sup> nerves are seldom involved by direct extension.

Occasionally the jugular foramen syndrome or the syndrome of Jackson is reported. These syndromes may involve the ninth, tenth, eleventh and twelfth cranial nerves. Symptoms would be referred to the tongue, palate, pharynx, larynx, sterno-cleido-mastoid or trapezius muscles.

The cranial nerves are usually involved extracranially but extension intracranially is not uncommon. The nerves passing through the sphenoid fissure are affected most frequently. The sixth nerve is the most commonly involved and the fifth next.

As the tumor develops in the nasal cavities, nasal blocking, purulent discharge, odor and hemorrhage are usually present.

In the later stage diplopia, blindness, proptosis, paresthesia of the face, dysphagia, aphonia and hoarseness may be present.

Inspection of the nasopharynx in the early stage may not reveal the presence of the tumor due to its hidden location, even though some of the cranial nerves are involved.

**Treatment**—Treatment consists in external irradiations by means of the roentgen rays or radium packs combined with postnasal application of radium to the lesion.

Coutard with his protracted fractional method of roentgenotherapy

<sup>1</sup>Jour. Am. Med. Assn. 79:10 (July 1) 1922.

<sup>2</sup>The author (H. C. B.) reported a case with a total destruction of the eighth nerve from a direct extension of a carcinoma of the nasopharynx. *Ann. Otol., Rhinol. and Laryngol.* 42:899 (September) 1932.

obtained three-year cures in 32 per cent of his cases. Other investigators report from 0 to 25 per cent five year cures.

The technic employed by Coutard consists of approximately 250 roentgens per hour for twenty five to thirty five hours of irradiation extending over approximately fifty days. This permits the total dose of radiation of approximately 8500 roentgens. The disadvantages of the method are the expense and length of time necessary to carry out the treatment.

The radium bomb or pack consists in the use of from 2 to 4 gm. of radium, highly screened, at a distance ranging from 6 to 15 cm. Sufficient time has not elapsed to determine the final results from this form of therapy.

Surgical removal is contraindicated especially in the anaplastic types.

### MALIGNANT NEOPLASMS OF THE TONSILS

**Etiology** — Schall<sup>1</sup> in reporting 230 patients with carcinoma of the tonsil seen at the Collins P. Huntington Memorial Hospital found the

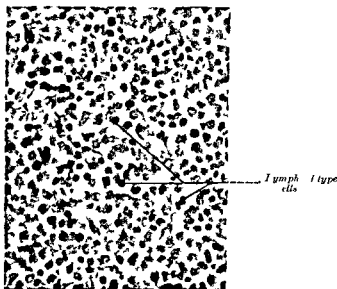


FIG. 355 — Lymphosarcoma of the lymphoblastic type ( $\times 400$ )

incidence of tonsillar involvement about 1 in every 100 cancer patients.

Carcinoma of the tonsil occurs in men in the vast majority of cases. It is usually found past middle life but may occur at any age. In a small percentage of cases a family history of cancer can be obtained. Tobacco may or may not be a factor in the etiology. When seen by the physician the disease as a rule is not limited to the tonsil but has extended to the adjacent structures or the lymph nodes.

<sup>1</sup> Annals Otol. Rhinol. and Laryngol. 43: 1047 (December) 1934.



**Pathology**—New and Childrey<sup>1</sup> in reporting on 357 tumors of the tonsil and pharynx seen at the Mayo Clinic found squamous-cell epithelioma in this situation almost five times more common than all the other malignant tumors. Most of them were of an active type of growth. Under squamous-cell epitheliomas they include endothelioma, bronchiogenic carcinoma, lympho-epithelioma and transitional-cell carcinoma.

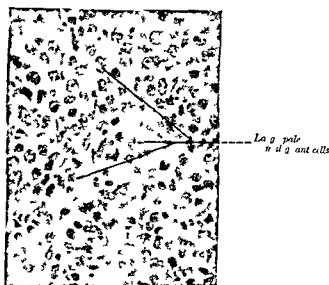


FIG. 356 Lymphosarcoma of the reticulo-endothelial type (X 400)

Sarcomas of the tonsils and pharynx are principally of the lymphosarcoma type. It is probable that many of the growths formerly called lymphosarcomas are lympho-epitheliomas. They are frequently primary in the tonsil. Lymphosarcoma of the tonsil is usually a dull reddish pink color with a rounded swollen contour or the surface may be grayish or nodular. Ulceration occurs late. The growths tend to become bulky and remain more or less limited for some time. In the pharynx lymphosarcomas may be pedunculated and movable or they may show diffuse swelling only.

There is a rather constant progressive secondary anemia but there may be a relative or absolute lymphocytosis. Increase of the polymorphonuclear leukocytes is sometimes seen.

On microscopic examination of a diseased node a complete replacement of the normal lymphoid structure by new cells is seen and this may involve either the lymphocytic or reticulo-endothelial elements. The cells of the first type resemble or are identical with lymphocytes whereas in the second case large pale cells with vesicular nuclei and a more open arrangement are found. The former type is known as a lymphocytoma and the latter as a reticulum-cell sarcoma.

Low grade epitheliomas are hard on palpation often whitish gray and of a dry glazed appearance. They ulcerate early with an irregular surface. Bleeding does not occur until late. The more active lesions (anaplastic) are softer and more rapidly growing. Infiltration of surrounding tissues occurs rather than large surface growths.

**Symptoms**—Symptoms and signs of malignant tumors of the tonsil as a rule attract little attention until the tumor has reached a considerable size. Enlarged cervical lymph nodes frequently are the first sign of trouble as the lymph node enlargement occurs fairly early. Occasionally no evidence of enlarged lymph nodes are found. The enlargement varies from small palpable to large masses in the neck. The nodes when present have a hard matted feel.

The later symptoms usually complained of are pain sensation of a growth in the throat bleeding hoarseness dysphagia dysphonia and ulceration.

**Differential Diagnosis**—These malignant lesions should be distinguished from simple enlargement of the tonsil Vincent's angina peritonsillar abscess adenocarcinoma of the mixed tumor type and benign tumors. The metastasis should be differentiated from tuberculosis syphilis goiter carotid body tumors and cysts.

## TREATMENT OF MALIGNANT NEOPLASMS OF THE PHARYNX MOUTH AND TONSIL

The selection of the proper treatment of malignancy should be based on the histologic grading in given types the gross appearance clinical course location and origin previous treatment and local or general conditions of the patient.

Definite types of tumors such as the transitional-cell carcinoma lympho-epithelioma and basal-cell carcinoma are very sensitive to irradiation. Round reticular-celled and lymphosarcomas are also sensitive. Adenocarcinomas and fibrosarcomas are relatively resistant.

Tumors with well matured cell forms with much stroma with hornification or other indication of specialization yield well to surgery or electrosurgery and often poorly to irradiation. Basal-cell carcinoma gives good results with either.

Very immature cell forms undifferentiated with little stroma approaching Grade 3 or 4 of Broder's system yield much better to radiotherapy. The great majority of epitheliomas of the pharynx nasopharynx posterior third of the tongue and tonsils are of the highly cellular undifferentiated type of transitional-cell tumors. They are highly radiosensitive and are best treated by irradiation.

Favorable sites for surgery or electrosurgery are the skin of the face ear nose and lips the maxillary sinuses palate alveolar ridge cheek epiglottis and anterior third of the tongue.

If a carcinoma of the anterior third of the tongue is localized wide excision by cautery or electrocoagulation supplemented with implantation of gold radon seeds is indicated. If the neoplasm is more extensive

the entire anterior portion of the tongue may be destroyed by diathermy treating lesions of the anterior portion of the tongue with a radium bomb containing 4 to 5 gm or with roentgen rays at 400 kv at a skin target distance of 100 to 110 cm has given good results without serious risk to the surrounding tissues

The cervical lymph nodes should be removed in all low grade (radio-resistant) carcinomas of the pharynx and tonsil if the prognosis of the local lesion warrants. If the epithelioma is Graded 3 or 4 or the nodes are from a lymphosarcoma they should be irradiated with radium packs or deep roentgen rays. Many observers prefer to use radium for the treatment of the local lesions and the roentgen ray for the external irradiation of the neck. The radium treatment may consist of radon implants in the form of seeds placed in such a manner that the lesion will receive uniform irradiation. If preferred the roentgen ray alone may be used after the technic of Coutard<sup>1</sup>

Coutard<sup>2</sup> reports five-year cures were obtained in tumors of the tonsillar region in 32 per cent (21 out of 65 cases). Tumors of the larynx 25 per cent (29 out of 114 cases) and tumors of the hypopharynx 11 per cent (23 out of 200 cases). Schall reports 18.4 per cent cures of carcinoma of the tonsils. Duff reports 20 per cent of five-year cure. These percentages compare favorably with those of Berven.<sup>3</sup>

**Coutard Technic** — For epitheliomas of the upper air passages Coutard uses voltage of about 200 kv with 5 milliamperes. The filter consists of 2 mm Zn + 3 mm Al. The focal skin distance varies from 40 to 60 cm. One lateral field on either side of the neck is usually employed sufficiently large to include the primary lesion and regional metastases. Doses recorded as r units on the skin of from 5000 to 8000 r are used. This represents the total surface dose usually given over all fields. The largest proportion of the dose is given on the side of the lesion.

If 150 r are given daily for fifty days a series of slight periodically returning reactions or inflammations of the mucous membrane and skin (radioepithelitis and radioepidermitis) occur. Coutard found they recur periodically after two four six and eight weeks on the mucous membrane and after four and eight weeks on the skin.

**Teleradium** — Cutler uses teleradium exposing the lesion to the gamma rays of radium (4 gm pack and 2 gm pack) twice daily on consecutive days without interruption for periods varying between twenty and sixty days or until the characteristic reaction is observed in the normal tissues surrounding the growth.

### MALIGNANT NEOPLASMS OF THE NASAL CAVITIES

Malignant tumors of the nasal cavities are rare and when present early invasion of one or more of the sinuses usually occurs.

**Symptoms** — Unilateral nasal obstruction and nasal bleeding are the most prominent symptoms. Early ulceration and bleeding may be

<sup>1</sup> Rad ophys et Rad otherape ( ) 541 et seq 1930 1932

<sup>2</sup> Laryngoscope 46 407 (June) 1936

<sup>3</sup> Acta Radiologica Supp x (1931)

noticed before nasal obstruction if the growth is small and of the anaplastic type with thin walled blood-vessels. If the tumor is the slower growing radiation resistant type (Grades 1 or 2 according to Broder's system) ulceration and bleeding may not occur until after the nasal stenosis is marked. The growth may be mistaken for a nasal polyp. It is most frequently found on the nasal septum or on the superior

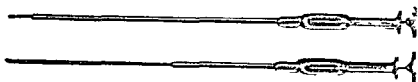


FIG 35\* —Loading slot instruments for implanting radium seeds. Small quantities of emanations in the form of seeds fixed in capillary tubes can be placed in some tumors and left there permanently (1 millicurie implant in each square centimeter of tumor mass). The seeds lose their intensity in about thirty days and are left as small sterile foreign bodies. They may slough out if embedded in an infected area. They are particularly suitable for superficial growths where a caustic effect is desired on the surface with no effect on the deeper structures. (Courtesy of The Radium Emanation Corp.)

lateral wall of the nasal passage. Erosion of bone as determined by roentgenograms indicates an extension of the growth beyond the nasal mucosa.

The diagnosis is confirmed by a biopsy taken from a non necrotic portion of the tumor.

**Treatment**—The treatment of malignant growths within the nasal cavity with little or no evidence of bone involvement is complete surgical removal if possible followed by irradiation.

**Surgical Treatment**—Havens and Thornell<sup>1</sup> advocate a fronto-ethmoid approach to the nasal cavity.

An incision, through the soft tissues and periosteum, is started just under the unshaven eyebrow 1 cm. lateral to its inner extremity and extended medially and then inferiorly midway between the middle dorsum of the nose and the inner canthus of the eye to end just below the free edge of the nasal bones.

The periosteum is elevated by means of a sharp periosteal elevator from the middle of the nose to the lacrimal bone, care being taken to avoid injury to the lacrimal sac. It may be necessary to elevate the lacrimal sac from its fossa and the periosteum from the orbital plate of the ethmoid if the growth has extended into any portion of the ethmoid sinus.

An incision is then made through the nasal mucosa along the lower free edge of the nasal bone and the mucous membrane elevated from the under surface of the nasal bone.

Portions of the nasal bone and nasal process of the maxillary and frontal bones are removed by means of rongeur forceps.<sup>1</sup> The nasal mucosa is then incised parallel to the skin incision creating an opening

<sup>1</sup> Arch. Otolaryngol. 40:396 (November) 1944.

into the nasal cavity through which the attachment and extent of the tumor can be determined. It may be necessary to open and examine the various sinuses if evidence of an extension of the growth is found.

After removal of the tumor the area of attachment is electrically coagulated and radium applied. The radium is held in place by gauze picking and subsequently removed through the nostril as the skin wound is closed.

### **MALIGNANT GROWTHS OF THE NASAL SINUSES**

All the nasal sinuses may be the site of a malignant growth either primary or by extension. A primary carcinoma is most commonly encountered in the maxillary sinus. The ethmoid, frontal and sphenoid sinuses in the order named are less frequently involved. The sinuses other than the maxillary are usually involved by secondary extension.

Sarcoma usually seen in younger individuals frequently follows an injury.

Malignant growths of the sinuses occur in males as a rule with a ratio of about seven males to one female.

Carcinoma of the antrum is not an uncommon disease. Ewing states that out of 1892 cases of cancer of all types 35 (1.84 per cent) involved the maxillary sinus. He recognizes the following types:

Papillary carcinoma, some of which are malignant transformation of papillomata.

Carcinoma of basal cell type. They are often designated as adenocystic epithelioma, endothelioma or cylindroma.

Squamous-cell carcinoma which arises by metaplasia from previously altered lining epithelium.

Cylindrical-cell carcinoma which forms a bulky tumor and is unusually malignant. It is adenocarcinomatous in type.

Round-cell carcinoma of atypical structure which is often designated as sarcoma.

Dental tumors which not infrequently develop in the antrum. They include the squamous and glandular types of adamantinoma.

**Symptoms**—The symptoms of early lesions are absent. A sense of fullness in the antrum and tumefaction of the soft tissue of the cheek may occur. A localized or radiating pain to the teeth may be present especially if the floor of the antrum is involved. Parathesia or anesthesia of the cheek follows involvement of the posterior or superior antral walls. If the hard palate or superior alveolus are invaded the teeth loosen and fall out but with little or no pain. Extensive ulceration with fistulation of the maxillary sinus may occur in the late stage. In the early stages transillumination, antral lavage and intranasal examination may show nothing. Roentgen examination with an opaque oil will frequently suggest the presence of a growth.

If the tumor originates from the ethmoids or around the orbital plate an early swelling around the eye or a persistent puffiness over the cheek may be present. Nasal obstructions is a later symptom. The growth bleeds easily on probing. Examination reveals a firm, vascular somewhat pinkish tumor covered with a bloody mucoid secretion as a rule.

If the growth invades the nasopharynx deafness trigeminal neuralgia or persistent otalgia with negative ear findings may be present.

Extension to the sphenoid sinus may involve the optic nerve resulting in defective vision on the affected side.

Watson<sup>1</sup> in reporting 127 cases of primary cancer originating in one of the paranasal sinuses seen at the New York Memorial Hospital found metastasis occurred in 29 per cent usually by a direct extension through the lymphatic pathways. Metastasis by way of the blood stream to the lungs bones liver spleen etc may occur but it is very infrequent.

**Idenocarcinoma**—An adenocarcinoma reproduces in a rough way the epithelial glandular tissue from which it arises (Fig 338).

The symptoms are those resulting from obstruction and pressure. Persistent pain in the cheek temporal region or forehead may be complained of. A polypoid vascular mass may be seen in the middle meatus which bleeds freely on touching. As the tumor develops the eye may bulge or the lacrimal duct become obstructed. A puffy swelling of the cheek below the eye is a late sign. Primary melanosarcoma is rare in the nose.

**Diagnosis**—The diagnosis of carcinoma of the sinuses is made from the symptoms and signs and from the biopsy.

The differential diagnosis should be made from trifacial neuralgia maxillary sinusitis dental diseases syphilis and aspergillosis of the antrum.

**Prognosis**—Watson reports a five year survival rate of 20.5 per cent. Malignant neoplasms of the nasal cavity secondarily involving the antrum usually give a higher percentage of cures than primary lesions of the antrum probably due to the earlier diagnosis and greater accessibility for treatment. The growth in the upper jaw rarely metastasizes unless the tumor has extended to the cheek.

Ohngren divides the antrum into four different malignancy planes. Malignant growths having their origin in the anterior and inferior portions of the sinus are considered to be less malignant than those originating in the posterior and superior portions. The close relationship of tumors in the latter position to the orbit and base of the brain may account for the discrepancy.

**Treatment**—The treatment of choice of malignant tumors of the antrum is diathermy and radium. A sublabial approach is made and the tumor removed by electrocautery is advocated by Ohlgren. If surgical diathermy is used the periphery of the growth should be destroyed first and then the tumor removed or destroyed with a button or needle electrode. The slough is then curetted away. Large amounts of radium are used later.

If exophthalmus is present and it is questionable whether or not the orbit is involved by the growth Schall<sup>2</sup> evertes the orbital contents

<sup>1</sup> Laryngoscope 52 72 (January) 1942

<sup>2</sup> Acta Otolaryngol. Supp. 19 1 1933

<sup>3</sup> Laryngoscope 53 240 (April) 1943

and enters the maxillary sinus from above and the ethmoid labyrinth from the side. Lesions originating in the superior alveolus or hard palate with extension into the maxillary sinus may be reached by resection of the superior alveolus and part or all of the hard palate. For extensive sinus involvement Schull uses a modified Moore incision starting opposite the inner canthus of the eye extending down the lateral wall of the nose around the ala to the mid line of the upper lip which is then divided in the mid line. An incision is then made through the buccal mucosa from the nasal spine to the last molar tooth. The entire cheek is turned back permitting removal of all or part of the maxilla if necessary.

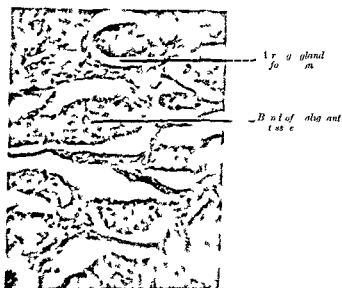


FIG. 358. Adenocarcinoma ( $\times 200$ ). Mitoses are numerous.

Röntgen and radium therapy in adequate dosage offer hope of cure in the radiosensitive types. It may be necessary to use some form of a surgical procedure to obtain a proper exposure of the growth so that irradiation may be used.

Teleoradium (teleo Curie therapy) is the use of the radium bomb in which large amounts (2 to 4 gm) of radium in lead containers are used.

The surgical removal of the growth consists in removing the lymph nodes ligating the external carotid if necessary and excising the lesion of the cheek by diathermy. However Quick<sup>1</sup> advocates the conservative treatment of the areas of the lymph node drainage of the neck rather than routine block dissection.

In inoperable cases opiates, alcohol injections of the fifth nerve or Gasserian ganglion or a resection of the posterior root may be necessary to alleviate the pain.

**Adenocystic Carcinoma**—Adenocystic carcinomas of the respiratory mucosa have been reported at various times in the literature as cylindromas, basal-cell carcinomas and endotheliomas. Bredlau<sup>1</sup> lists them as varieties of basal-cell carcinoma subdivided into solid, cystic, adenoid or adenocystic as advocated by Krompecher.

**Pathology**—Basaloma, cylindroma, endothelioma and basal-cell carcinoma are most frequently found in the respiratory passages and the skin about the face. They present histologically similar lesions and pursue about the same clinical course.

The neoplasm is usually a red fleshy mass more or less firm in consistency which at times may appear polypoid. It bleeds readily. The growths may destroy the surrounding bone. Metastases are rare.

Histologically it presents a picture of cell nests surrounded by a connective tissue stroma. The cell nests frequently show a central lumen containing a pink staining material and sometimes cellular debris.

**Symptoms**—They are characterized clinically by slow growth, local progression, lack of metastases, prompt regression to irradiation and recurrence after incomplete excision.

The symptoms are dependent upon the site of involvement. The most frequent area involved is the maxillary sinus. Bredlau found the maxillary sinus involved in 68 per cent of the cases. The most common complaint is nasal blockage. Repeated attacks of epistaxis are frequent. Visual disturbances are sometimes noted. Headache and pain in the cheek are common complaints.

Objectively a mass in the nostril with a bulging or swelling of the cheek and palate on the affected side can be observed. Transillumination and roentgen rays show cloudiness of the affected side. Other regions involved such as the nose, sphenoid, palate, base of tongue, epiglottis, larynx and trachea would give symptoms referable to the regions affected.

**Treatment**—They are relatively radiosensitive and best treated by cauterization and adequate irradiation.

**Ollier's Operation**—Ollier's operation may be indicated in some cases for better access to the nasal cavities. In other instances Moore's lateral rhinotomy would be better. Instead of separating the nose from above it may be done sublabially from below.

Ollier's operation is performed under general anesthesia with the head of the patient hanging over the end of the table in Rose's position. Postnasal trunks should be introduced to prevent entrance of blood into the nasopharynx and larynx. An incision extending from the left ala of the nose upward over the bridge and thence downward to the right ala should be made through the cutaneous tissue (Fig. 370). A Gigli saw should then be placed at the bridge of the nose and all the bony structures along the cutaneous incision severed.

The nose, thus temporarily resected, is then turned downward over the mouth. This having been done the growth should be enucleated by blunt dissection if possible or if this cannot be done it should be destroyed by diathermy. Hemorrhage may be considerable hence the

<sup>1</sup> *Annals Otol. Rhinol. and Laryngol.* 45: 894 (September) 1936.



postnasal tampons introduced before beginning the operation serve as bases against which strips of gauze may be packed to check it.

Having removed the tumor the incision should be closed by sutures and the tip of the nose raised into position and fixed with adhesive strips. The stitches should be removed on the fifth day. The nasal wound should be packed with gauze impregnated with bismuth or vaseline. The intranasal dressing should be renewed daily.

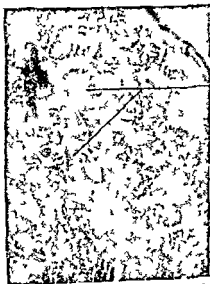
Radium and roentgen ray can be used later as indicated.



FIG. 359.—Operation for exposing the nasal cavity for operative purposes.



FIG. 360.—Lateral view of the Operation.



— Cord of malignant tissue

FIG. 361. Basal-cell carcinoma of the nose ( $\times 80$ ). Solid masses of darkly staining cells can be seen extending down from the epidermis. These cords of cells contain no cell nests, cornification, and usually no mitotic figures.

## MALIGNANT NEOPLASMS OF THE EXTERNAL NOSE

Malignant neoplasms of the skin of the nose may be divided into two main groups. Basal-cell carcinoma and prickle-cell carcinoma or epithelioma. The basal-cell carcinoma is characterized by the usual pearly border and slow development without metastasis. This is commonly called *rodent ulcer*. The second type of neoplasm or carcinoma spino-cellulare is much less commonly observed. It is commonly called *prickle-cell carcinoma* or *epithelioma*.

Basal-cell carcinoma may appear upon any skin surface but occurs most commonly on the nose and forehead. The lesion is usually solitary. The basal cells of the epithelium grow down into the stroma.

According to Barlow. The rodent ulcer appears first as an elevated whitish nodule or patch covered with scales which tend to drop off. It is essentially a slow process but sooner or later an ulcer forms with very little tendency to heal at the base. It is usually superficial but in time it may become very extensive and even erode bone and cartilage. A crust may form over the dirty necrotic base and ulceration develops when the cells become so far removed from their source of nutriment that necrosis takes place. Additional nutrition is then supplied to the surviving cells and the lesion may extend rapidly. The extension of the lesion may produce distressing deformities of the whole region as when the nose, cheek and upper lip are destroyed.

According to McFarland if the growth is characterized by a disposition of the multiplying cells to complete the natural differentiation and pass through the prickle-cell stage to that of true keratinization we have the carcinoma acanthous or spino-cellulare.

The treatment of carcinoma spino-cellulare consists in wide excision either by knife or cutting cautery. If the denuded area is large skin grafting may be resorted to later. Radium and roentgen ray may be used later as indicated. Good results may be had in many instances from radium or roentgen ray alone.

## PART IV

### THE EAR

#### CHAPTER XXXVII

### THE CLINICAL ANATOMY AND PHYSIOLOGY OF THE EAR

THE organ of hearing is divisible into The external ear the middle ear and the internal ear

#### THE EXTERNAL EAR

From a clinical point of view the auricle is of interest on account of the destructive inflammatory processes which attack its cartilaginous framework and the perichondrium covering it Perichondritis and

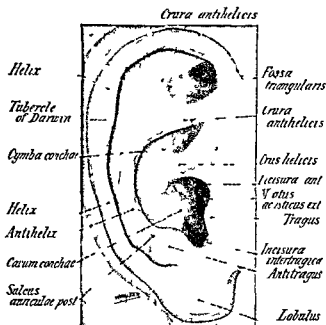


FIG 360 —The auricle

chondritis of the auricle from traumatism or following the mastoid operation occasionally occurs In performing the plastic operation upon the canal in the radical mastoid operation the cartilage of the auricle is included, and is, at times followed by an infection of the perichondrium and cartilage

The external auditory canal is divisible into a cartilaginous and an osseous portion The cartilaginous portion of the canal (the auricular

extension) is attached to the osseous or deeper portion by bands of fibrous tissue. The length of the canal averages from 35 to 38 mm. It runs forward and inward in an oblique direction and presents a curve which is higher near the middle of its length than at either extremity. Throughout its entire extent it is lined by cutis closely adherent to the underlying tissue. The superior and posterior walls of the cartilaginous canal are thinner than the anterior and inferior walls. The inferior wall extends deeper along the floor of the canal than the other walls and is known as the *processus triangularis*. The anterior wall of the cartilaginous canal is crossed by two or three fissures which are filled with connective tissue and a few muscle fibers. These fissures called the fissures of Santorini render the auricle more movable. A deep abscess of the parotid gland may find an outlet through these fissures.

In the new born the canal is fibrous throughout its entire length and its walls are collapsed and in apposition. Bone salts are gradually deposited and the canal assumes its patulous condition.

The sebaceous glands are limited to the cartilaginous portion of the canal hence furunculo is confined to this area.

Four cranial nerves give branches to the external ear. The auriculo-temporal branch of the third division of the fifth sensory branches from the geniculate ganglion of the facial. Jacobson's nerve from the ninth Arnold's nerve from the tenth or vagus nerve.

### THE MIDDLE EAR

The middle ear embraces Tympanic membrane or drum head eustachian tube the tympanic cavity and its contents (the ossicles ligaments muscles etc.), antrum mastoid cells.

**The Tympanic Membrane**—The eardrum is stretched across the inner end of the external auditory canal. It is elastic enough to undergo considerable movement when the air in the canal is alternately condensed and rarefied with Siegle's otoscope. At the inner end of the external auditory canal is an incomplete bony ring the *annulus tympanicus*. On the surface of the annulus there is a groove the *sulcus tympanicus* to which the ear-drum is securely attached by an extension of the periosteum of the bone and the middle fibrous layer of the drum. The *annulus tympanicus* does not extend completely around the canal opening but is absent at the upper portion the *Rivian segment*. The part of the membrane attached to the annulus is known as the *pars tensa* or the *membrana tensa*.

The part attached to the Rivian segment is not stretched but is loosely drawn and is known as *Shrapnell's membrane* the *pars flaccida* or the *membrana flaccida*. This portion of the membrane forms the outer wall of Prussak's space while the *pars tensa* forms the lower portion of the outer wall of the tympanic or middle-ear cavity (Fig 363).

The tympanic membrane is a composite membrane of three layers. The outer one being a reflection of the skin of the canal made up of

layers of flat epithelium with cylindrical cells in the deepest layers the middle one being fibrous tissue consisting of an outer radiating and an inner circular layer of fibrous tissue poor in elastic fibers and the inner a reflection of the mucous membrane of the middle ear consisting of simple cuboidal epithelium. The handle of the malleus is embedded within these structures.

In that portion of the drum membrane known as the *pars flaccida* or Shrapnell's membrane a small concentric shaped field situated between the short process and the Rivinian segment the middle or fibrous layer is absent. The outer skin layer and the inner mucous membrane layer form the *pars flaccida*. The remaining portion of the drum membrane or the *pars tensa* is made up of the three layers as given above.

The size of the drum membrane is about 8 mm wide 9 mm high and about  $\frac{1}{10}$  mm thick. It is not a plane surface but indrawn like a shallow funnel the umbo forming the apex. It is inclined in both vertical and horizontal planes at about 45 degrees so that the superior and posterior quadrants are nearer the observer than the anterior and inferior quadrants. In infants this inclination is more marked so that the membrane more nearly approaches the horizontal.

The tympanic membrane presents certain landmarks by which its presence is recognized. A prominent one is the *short process* of the malleus which is seen as a more or less prominent grayish white point in the upper and anterior portion. Extending downward and backward from the short process to the middle of the drum membrane is the *handle of the malleus* or the *manubrium*. At the tip of the manubrium is a flattened area called the *umbo*. Upon

looking at the drum membrane by means of reflected light a cone-shaped *light reflex* can be seen having its apex at the umbo and extending downward and forward. From the short process of the malleus extend two folds in the membrane. One passing forward is called the *anterior fold* and the other much longer is called the *posterior fold*. These folds form the lower boundary of Shrapnell's membrane.

In addition to the landmarks just mentioned one is sometimes able to see through the membrane some of the contents of the tympanum. Of these the most prominent is the long process of the incus. This is



FIG 363 Coronal section through the tympanum. a extremity of the upper meatus d tegmen tympani e e at external port on internal port on f malleus and superior ligamentum mallei g incus h stapes within the fenestra vestibuli i promontory k Prussak's space m hypotympanic recess (cellar) l scar in the lower half of the drumhead in apposition with the promontory n incudo-stapedial junction (after Bruhl Poltzer)

seen projecting downward to a point about the center of the posterior superior quadrant. From the tip of the long process of the incus extending inward the shadow of the stapes can sometimes be seen. At times the chorda tympani nerve is observed passing from behind forward across the tympanum just below the level of the posterior fold.

For convenience of describing the drum membrane is divided into quadrants. A line drawn through the short process and the umbo and another line at right angles to this and passing through the umbo will divide the drum membrane into four parts: an anterior-superior and anterior-inferior, a posterior-superior and a posterior-inferior quadrant (Fig. 364). Behind the anterior-superior quadrant lies the opening of the eustachian tube, the anterior mucous pouch of the drum and the canal for the tensor tympani muscle. Behind the anterior-inferior quadrant lies the carotid canal. Behind the posterior-superior quadrant is situated the stapes, the chorda tympani nerve, the long process of the incus, the posterior mucous pocket of the drum membrane and the pyramid containing the stapedius muscle. Behind the posterior-inferior quadrant lies the niche of the round window.

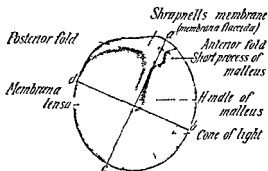


FIG. 364. The right tympanic membrane. *a b* anterior superior quadrant *b c* anterior inferior quadrant *c d* posterior inferior quadrant *d a* posterior superior quadrant.

**The Eustachian Tube**—The most common avenue of approach of infection to the middle-ear cavity is through the eustachian tube. It is through this channel that nearly all middle-ear diseases invade the middle-ear cavity. The tube is about 36 mm long, the pharyngeal opening being about 15 mm lower than the tympanic opening. The tympanic opening corresponds to the antero-superior quadrant of the eardrum; hence it is not in the most dependent portion of the cavity. This does not interfere with drainage under normal conditions, as the cilia of the epithelium of the tympanic cavity sweep the secretions to the opening of the tube and through it to its pharyngeal opening.

The tympanic end of the tube has an osseous framework and is about 12 mm long. The pharyngeal end of the tube has a cartilaginous and membranous framework and is about 24 mm long. The osseous portion is in relation above with the canal for the tensor tympani muscle and internally with the canal for the internal carotid artery.

The membrano-cartilaginous portion is made up of an inner membranous part and an outer cartilaginous wall. The tube is trumpet shaped at both extremities and is narrowest at the junction of the osseous and cartilaginous portion. This is known as the isthmus. The tube is lined with ciliated columnar epithelium with many goblet cells. The cilia carry the secretions toward the pharyngeal orifice.

Under ordinary conditions the membranous walls of the tube are in a state of collapse and only open when certain palatal muscles are contracted. Yawning and swallowing cause these muscles to contract and air is thus admitted into the tympanic cavity.

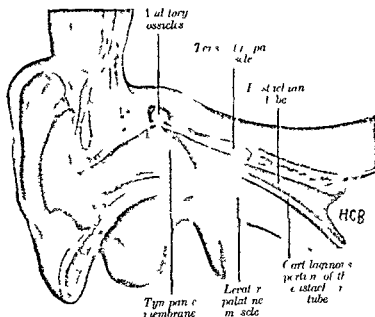


FIG. 36a.—Anatomical relation of the eustachian tube.

Three muscles act upon the tube: (1) the levator palatini which arises from the under side of the tip of the petrous bone and from the lower and outer surface of the eustachian cartilage and is inserted into the soft palate; (2) the tensor veli palatini which arises from the sphenoid fossa of the sphenoid from the angular spine of the sphenoid and from the anterior portion of the lateral membranous wall of the eustachian tube. This muscle is attached to the anterior surface of the soft palate; (3) the salpingopharyngeus which arises from the cartilaginous portion of the tube is inserted into the postpharyngeal wall.

The levator veli palatini is supplied by a pharyngeal branch (plexus) of the vagus; a branch of the facial. The tensor veli palatini is supplied by the mandibular division of the fifth nerve. These muscles elevate the soft palate and assist in approximating it against the posterior wall of the pharynx in the act of swallowing. As the superior ends of the muscles are attached to the cartilaginous lip and to the membranous portion of the tube and the inferior end to the soft palate it is obvious that the

contraction of the muscles will produce a two-fold result—namely the pharyngeal orifice of the tube is opened and the soft palate is elevated.

The action of the tensor and levator veli palatini muscles is so intimately associated with that of the muscles of the palate and pharynx that it is somewhat difficult to estimate the influence of the other muscles on the patency of the tubes. The pharyngopalatinus (posterior pillar of the fauces) has its upper attachment in the soft palate and it contracts during deglutition and thus indirectly exerts a tensile action upon the tubal muscles. In inflammatory processes involving the adenoid tonsils and faucal pillars the swollen condition of the palatopharyngeus muscle indirectly interferes with the action of the tubal muscles.

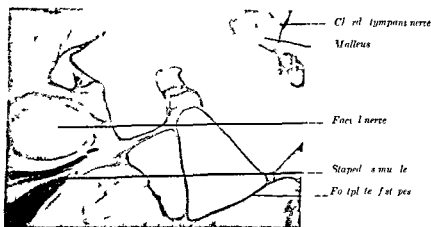


FIG. 366.—Section through the middle ear showing various structures (X 15).

The anterior wall of the pharyngeal end of the tube is membranous while the upper and posterior walls are cartilaginous. The tensor and levator veli palatini muscles are attached to the membranous portion of the tube; hence when they contract the tube is opened to its isthmus.

This permits of the interchange of air between the pharynx and the middle ear and maintains an equilibrium of pressure on the inner and outer surfaces of the membrana tympani.

The pharyngeal end of the tubal cartilage (posterior and superior walls) forms a projecting lip or tubal prominence (torus tubarius) on the lateral wall of the nasopharynx. Just behind this is a groove known as Rosenmüller's fossa. The fossa and tubal prominence are the landmarks used in the introduction of the eustachian catheter.

**The Tympanic Cavity, Tympanum, Cavum Tympani**—The tympanic cavity is the space between the tympanic orifice of the eustachian tube and the mastoid antrum. It may be described as a flattened cylinder and is divided into three parts, namely:

1. The epitympanic space (vault attic) or that part lying above the level of the upper margin of the membrana tensa.
2. The atrium or that portion of the tympanic cavity situated between the levels of the upper and lower margin of the membrana tensa.



3 The hypotympanic space or that portion of the middle ear cavity lying below the inferior margin of the drum membrane

The mucous membrane lining the tympanum is pale slightly vascular and covered for the most part with low columnar ciliated epithelium. Over the pyramidal eminence, ossicles and tympanic membrane it possesses a flattened non-ciliated epithelium. The mucous membrane covers the walls and also invests the ossicles, ligaments of the ossicles, tendons of the stapedius and tensor tympani muscles, and forms certain folds which are described later. It is continuous with the mucous membrane of the mastoid cells and the eustachian tube. The wave-like motion of the cilia carries the secretion to the eustachian tube.

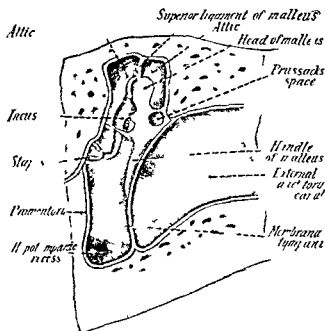


FIG. 367.—Schematic drawing showing a coronal section through the tympanic cavity.

The mucous membrane of the tympanum forms several vascular folds which extend from the roof of the tympanum to the ossicles, muscles and nerves. Pouch-like cavities are thus produced. One of these, Prussak's space, is well marked and lies between the membrana flaccida and the neck of the malleus. Two other pouches, the anterior and posterior spaces of Frotsch, are formed by the mucous membrane investing the chorda tympani nerve, one being anterior to the manubrium of the malleus and the other posterior.

The upper wall (tegmen tympani) of the tympanic cavity forms a portion of the floor of the middle fossa of the cranial cavity, the outer wall is composed of the eardrum and in its upper portion (outer wall of the attic) of bone. The wedge of bone forming the outer wall of the attic should be removed in the radical mastoid operation to expose this space to inspection and treatment during and after the operation.

The inner wall of the tympanic cavity is contiguous to the outer wall of the cochlea and vestibule the posterior wall separates the tympanic cavity from the antrum and mastoid cells the anterior wall is very thin and covers the internal carotid artery and the lower wall separates the tympanic cavity from the jugular bulb The facial nerve runs across the upper and posterior wall and is usually enclosed in a bony covering though numerous instances are on record in which the bony covering was absent

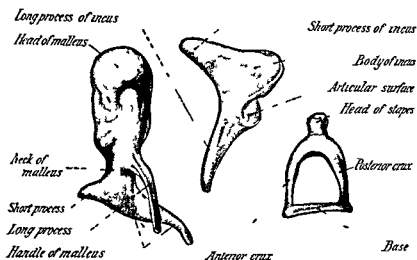


FIG. 368 The ossicles ( $\times 8$ ) (after Spalteholz)

**Contents of the Tympanic Cavity**—The tympanic cavity contains the chain of ossicles ligaments tympanic muscles and the chorda tympani nerve

**The Ossicles**—The ossicles (Fig. 368) are three in number the malleus incus and stapes They form a chain connecting the membrana tympani with the oval window of the labyrinth The malleus the largest of the three presents a head which lies in the vault behind the membrana flaccida and a handle (manubrium mallei) which is attached to the inner surface of the drum membrane Connecting the head and the handle of the malleus is a constricted portion called the neck Anteriorly the neck presents a long process (processus gracilis) The short process extends outwardly from the base of the handle

The incus presents a body and two processes The body of the incus occupies the posterior portion of the vault The anterior surface of the body of the incus articulates with the head of the malleus The short process of the incus extends backwards from the body and articulates with a small facet on the posterior wall of the vault just below the aditus ad antrum The long process of the incus extends downward and

backward and articulates with the stapes. This process is nearly parallel with the handle of the malleus.

The stapes or stirrup presents a foot plate which is attached to the oval window by an annular ligament. Two crura or arms arise and unite forming a neck. Above the neck is a small enlargement called the head.

Druss<sup>1</sup> in examining the normal histology of the articulations of the malleus, incus and stapes found they consist of four layers in each ossicle, three of which are more or less constant while the fourth is variable: (a) bony layer, (b) calcified cartilage layer, (c) hyaline cartilage, and (d) terminal layer (resembling fibrocartilage, connective tissue, endothelium, etc.). The articulation in his opinion is not that of a real joint in the true sense of the word, but is rather a type of symphysis between the two ossicles.

Five ligaments support the ossicles in the tympanic cavity, namely, anterior, superior and lateral ligaments of the malleus, the superior ligament of the incus and the ligament of the short process of the incus. In addition the foot plate of the stapes is attached to the oval window by the annular ligament. A loose capsular ligament binds together the incus and malleus and another capsular ligament encloses the incudo-stapedial articulation. Folds of mucous membrane cover the ossicles and ligaments forming pouches or pockets. Three of these pouches, the anterior pouch, the posterior pouch and Prussak's space are in contact with the drum membrane.

Two muscles are located within the tympanic cavity, namely, the stapedius muscle, innervated by the seventh cranial nerve, which arises from the pyramidal bony excrescence on the posterior tympanic wall and is inserted in the neck of the stapes, and the tensor tympani muscle, innervated by the fifth cranial nerve, which arises in a semicanal just above the osseous portion of the eustachian canal and from the upper wall of the cartilaginous portion of the eustachian tube. The tendon of this muscle after passing backward and upward across the inner wall of the tympanum within a special bony canal emerges in front of the oval window. It then curves around the processus cochleariformis, then crosses to the lateral wall to be inserted into the inner and anterior surfaces of the handle of the malleus.

The *chorda tympani nerve* enters the tympanic cavity through a small canal in the posterior region and passes between the handle of the malleus and the long process of the incus to an exit (canal of Huguer) at the inner extremity of the glaserian fissure. It passes down between the pterygoid muscles to join the lingual nerve.

**Walls of the Tympanum**—The superior wall, the tegmen tympani, is a thin plate of bone forming a portion of the middle fossa of the skull. It is continued backward to form the roof of the tympanic antrum and prolonged forward to form the roof of the canal containing the tensor tympani muscle. This plate of bone is frequently very thin and

or loudness of sound at each pitch at which the normal ear experiences a sensation of feeling as distinguished from hearing and above which a sensation of acute pain is experienced. The sound intensity necessary to produce this threshold of feeling is taken as a practical upper intensity limit of hearing. Since there are no definite lower or upper limits of pitch which are incapable of stimulating the auditory nerve this sensation of feeling may be taken as the upper and lower pitch values at which a sound produces on the normal ear a sensation as much of feeling as of hearing.

The limits of sensation just described may be taken as defining the confines of the normal auditory sense as every sound possible to perceive in a practicable way by the auditory sense or which is in any way serviceable must be included within them.

The abnormal auditory sense may be described with reference to the normal in the same terms and, if properly represented a comparison may be made showing the character of the differences.

The differences in intensity are measured in sensation units (s u), decibels (d b) or in percentage hearing. The sensation unit corresponds to approximately the smallest fractional change in intensity perceptible to a person of normal hearing. The decibel is a unit of relative intensity. It is the smallest increase in sound intensity appreciable by the normal ear. It is a logarithmic unit in which 10 decibels would correspond to a tenfold increase in sound intensity, 20 to a hundredfold 30 to a thousandfold, etc.

Various aids have been used to help determine the type location and degree of the hearing defect. The most common and probably the most practicable from the patient's standpoint is the whispered or spoken voice. However, this falls far short in determining the qualitative type of lesion as the primary voice sounds are limited to a range of from about 300 to 3000 cycles per second. Overtones may go higher. The male voice (fundamental and overtones) ranges from about 80 to 7800 frequencies while the female voice ranges from about 160 to 10 250 cycles. Adequate hearing for the conversational voice is possible however in individuals who have retained fairly good hearing for the frequencies 512 to 2048. The voice is also unsatisfactory from a more exact quantitative standpoint because of the variations in the voice of the examiner at different times or places or under different conditions.

The tuning forks are a valuable aid and probably will not be supplanted by other forms of testing at least they should not be at the present time. The forks used in otologic practice are usually made in a range from 16 d v to 4096 d v and unless supplemented by the whistles or the monochord are inadequate for the high tones. They are also useless for testing the hearing of those who have impairments of hearing of 50 per cent or more as a person with that degree of deafness would not hear the forks.

Tests for hearing by means of the voice watch or tuning forks are essentially qualitative and as usually given are inadequate for more exact tests of hearing. It is difficult to express the results obtained by

such tests in terms of decibels (d b), sensation units (s u) or percentage hearing

The audiometer is more satisfactory for testing the hearing in the higher tone ranges than the tuning forks or the Galton whistle. It is the best means for determining defects or "islands of deafness." The diagnosis of the cause of deafness can often be made more satisfactorily by means of the tuning forks. As a means for measuring the actual handicap from a defect in hearing the audiometer or forks are of limited value, as the voice itself is the only test that has much practical importance to the patient. As a means for measuring the progress or decline of deafness the audiometer (testing all the tone range) is of much value, especially as a method of precision over other methods.

**Air Conduction**—The intent in measurement of air conduction, as well as bone conduction, is to determine at a given pitch or frequency the faintest sound a person under test can hear.

The determination of threshold acuity is made more quickly and accurately with an audiometer than with tuning forks as the audiometer has a greater range of both frequency and intensity than do the forks.

Air conduction tests alone by means of forks or the audiometer do not permit of an accurate diagnosis regarding the location of conductive lesions. The same thing may be said of bone conduction tests in diagnosing nerve lesions, however by utilizing both air and bone conduction, combined with the otoscopic and fork examinations, the otologist may fix the location of the lesions with reasonable accuracy.

**Bone Conduction**—Bone conduction is the ability to hear a note or a noise when the activated source of sound is placed upon the head. Very little is known about hearing by bone conduction. Most observers favor the opinion that the important pathway to the inner ear is an osseous rather than an osseotympanic one. In any event the sound is perceived only when the nerve impulses reach the cerebral cortex.

The three methods usually used to test bone conduction are (1) the tuning fork, (2) the monochord and (3) the audiometer with a bone conduction attachment. The watch and acoumeter are used at times but are unsatisfactory. Tuning forks of medium low frequency (128 to 512 cycles) are the most satisfactory to use. The normal ear hears the sound by bone conduction (128 cycles fork) about one-half the length of time that it is heard through air conduction.

The bone conduction test may be altered by skull resonance as influenced by asymmetry of the skull, density of the bone or various conditions of the sinuses. For frequencies below 2000 cycles the hearing by bone conduction is influenced to some extent by lesions of the conduction apparatus but does not seem to be influenced appreciably for frequencies above 2000. In the latter event a direct test of the cochlea is made. The difference between the air conduction and the bone conduction gives the conductive loss. However, the hearing acuity for tones by bone conduction should be measured throughout the range of hearing. Knudsen and Jones have reported prolonged bone conduction is rarely observed in a sound-proof room.

In a pure conductive type of deafness the bone conduction should be normal for all tones as the cochlea would have normal acuity. In this type of deafness the loss of hearing by air conduction would be greater for the tones of low frequency.

In a purely perceptive type of deafness the curve for air conduction and bone conduction would be about the same if the defect is in the cochlea. The loss of hearing by both bone and air conduction would be greater for the tones of high frequency in a perceptive deafness.

The Committee<sup>1</sup> of the American Otological Society on the methods of testing the hearing by bone conduction recommends that all otologists routinely use the technic of alternate placements of the fork. The alternate placements at regular intervals begins immediately after the fork is struck and continues until it ceases to be heard by either air or bone conduction which ever is the more acute. The stem of the fork is held against the head for two out of each five seconds. The total cycle of each alternation should not exceed five seconds for the lower frequencies and three seconds for the high frequencies.

**Audiometer**—The importance of accurately determining the acuity and quality of hearing in delicate functional and diagnostic examinations has long been recognized. As a result several different types of audiometers capable of meeting a wide range of requirements have been developed.

Accurate tests may be made not only by trained otologists but also by office assistants or nurses.

The data obtained may be recorded by means of graphs which are easily interpreted and make possible a tentative diagnosis even before making a fully complete examination of the patient. The graphs visualize to the physician the defects in hearing and give a partial indication of the type of disease causing the deafness. Comparison of sequent charts demonstrates improvement in function or the contrary. The audiometer makes it possible to produce graphs of value in medicolegal cases.

The audiometer consists of three essential parts viz a frequency oscillator, an attenuation potentiometer which is a device for regulating the volume of sound and a receiver. It is acoustically a generator of approximately pure tones which may be varied both in pitch and intensity. The oscillator has a frequency range which in most instruments extends from 100 to 10 000 double vibrations or cycles per second. In some audiometers this range is divided into eight or more steps of frequency, 64 128 256 512 1024 2048 4096 and 8192 cycles per second. In others the frequencies are continuous.

The attenuation potentiometer consists of a resistance network connected to a switch which serves to vary the output of the oscillator. An attachment for testing bone conduction with or without masking the opposite ear is present on most instruments.

**Method of Testing Air Conduction**—To determine at each pitch the faintest sound the person being tested can hear the usual procedure is to begin with an intensity which is clearly audible. Upon hearing this

<sup>1</sup> Ann. Otol. Rhinol. and Laryngol. 45: 800 (September) 1936.

tone, the patient presses his signalling button, lighting the signal lamp. The potentiometer pointer is then slowly turned toward zero until the patient no longer hears the tone. He then signals this fact by releasing the button, extinguishing the lamp. Thus the exact measurement of the patient's hearing at the particular pitch is obtained. Frequent use of the tone interrupter throughout the test will check the accuracy of the patient's signals.

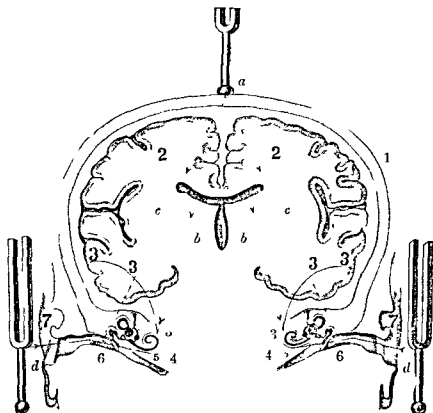


FIG. 378 — Air and bone conduction (schematic). 1 cranium 2 cerebrum 3 auditory nerve going to temporal lobe 4 labyrinth 5 tympanum and ossicles 6 auditory meatus 7 pinnae *a* tuning fork placed on the vertex *ab* osteal bone conduction *ac* cranio-tympanic bone conduction *d* tuning fork held in front of the ear *dc* air conduction (after Bruhl Politzer)

**Method of Testing Bone Conduction** — The same method of testing is used for both air conduction and bone conduction measurements. The bone conduction receiver is placed over the mastoid of the ear under test and measurements noted. The bone conduction receiver permits testing at frequencies considerably higher than are possible with tuning forks.

The lower limit for bone conduction testing is usually about 250 cycles per second inasmuch as a sensation of feeling as well as hearing is generally experienced at frequencies below 250 cycles.

**Masking Method of Testing Bone Conduction** — In the ordinary bone conduction test a fork is placed on the mastoid region and the entire

skull is vibrated. If the ears are of equal acuity the sound will be sensed in the ear close to the fork, but if the hearing in the opposite ear is better the sound will be perceived first in that ear. Consequently the test may not show the bone conduction loss for the deaf ear, but of the opposite or better ear.

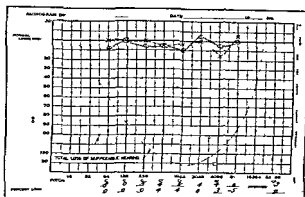


FIG. 39—Audiogram showing a plot usually associated with normal hearing.

The method of eliminating perception in the better ear is accomplished by introducing a so-called masking tone in that ear using the same or different frequencies as the test tone. The test tone may be intermittent so that its presence can be detected easily.

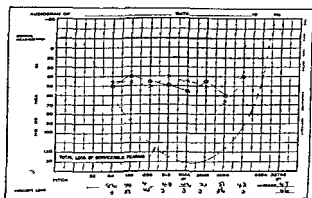


FIG. 380—Audiogram showing a plot usually associated with mixed deafness.

When making an audiometric bone conduction test by the masking method the bone conduction receiver is placed in contact with the mastoid bone and the air conduction receiver or other masking device is held to the opposite ear. The minimum intensity at which the interrupted tone is perceptible represents the bone conduction sensitivity or hearing loss for the ear under test.

In masking the hearing of the better ear while the poorer one is



being tested, an ordinary electric buzzer connected to a telephone receiver, as used by Jones and Knudsen in their audio-amplifier is satisfactory. The buzzer must not be so loud as to mask the hearing in the opposite ear. In many instances a stream of air from a compressed air tank, directed against the auricle or the external auditory meatus is a satisfactory and simple method of masking the non-tested ear. The help of an assistant is necessary in this latter method.

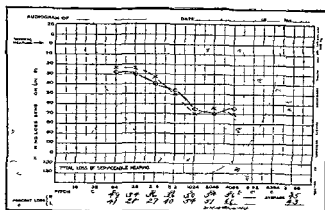


FIG. 381 — Audiogram showing a plot usually associated with nerve deafness

*Audiogram* — The audiogram is a chart so arranged that the results of measurements obtained by means of the audiometer or other instruments producing tones of known pitch and intensity can be plotted.

On the audiogram the entire region of normal hearing is represented

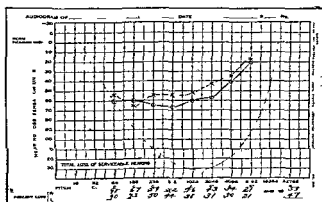
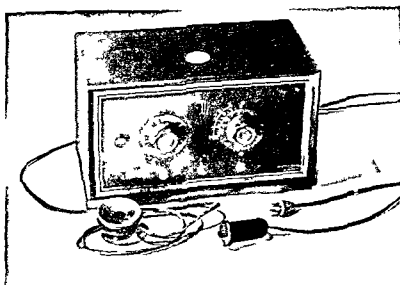
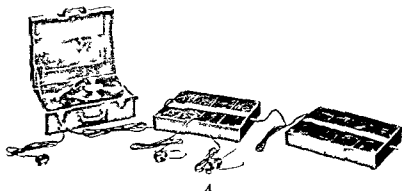


FIG. 382 — Audiogram showing a plot usually associated with obstructive deafness

the threshold of hearing being designated as "Normal Hearing," and the threshold of feeling, as "Total Loss of Serviceable Hearing." On this chart may be recorded audiometric measurements which show at a glance the extent and character of the hearing deficiencies. The horizontal scale represents pitch, corresponding to divisions of the

musical scale. The vertical scale indicates the intensity of sound in sensation units or decibels. The readings obtained with the audiometer give the threshold of hearing for the patient, comparable with the threshold of normal hearing represented on the audiogram by the line marked normal hearing. The result is a graphic presentation of the state of the patient's hearing which is roughly comparable with previous or subsequent audiograms of the same patient or of others.



B

F c 383 — A The 4-A audiometer and receivers for group testing. B 6-A audiometer for individual testing. (Courtesy of Western Electric Company.)

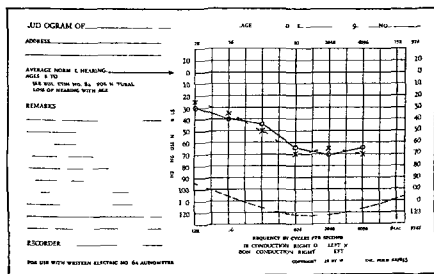
**Group Testing** — Pupils who lag behind others in classroom work have long been a problem to their teachers. The causes for the lagging naturally differ. It is only in recent years that the increasing interest in physical examinations and their use has proved that the lagging often results from physical defects and variations. Subnormality in

acuity of hearing in many cases accounts for apparent inattentiveness and lack of ability to understand instruction.

With the realization of this condition and the use of physical examinations to discover those with defective hearing, a need has grown for an instrument that will measure acuity of hearing accurately and quickly, and also use the same standard of measurement for all examined.

Figure 384-A shows three data sheets for use with 4-A audiometers. Each sheet contains a table for recording hearing data across various frequencies and intensities, with handwritten entries.

A



B

FIG. 384.—A Data sheets for use with 4-A audiometers B chart for use with 6-A audiometer

In addition it is obviously desirable to avoid the errors possible when a hasty whisper or watch-tick test is given for this purpose.

The 4-A audiometer (Western Electric) was designed to meet these conditions. This instrument is essentially a phonograph to which has been added telephonic apparatus so that the sounds produced in the

phonograph are transmitted to the ears of those under examination. With this instrument it is possible to transmit sounds to the listener's ears with a great degree of uniformity.

It affords a reproduction of conversational speech embodying a wide pitch range. It facilitates the detection of children who are deaf only in one ear, but who by special concentration of attention might pass other tests unnoticed.

The 4-A audiometer can be arranged so that one person alone or as many as forty persons simultaneously can be tested under the same conditions.

The pupil by writing down the numbers heard indicates definitely where in the range of sound the ear recognized sounds and is able to interpret them correctly.

The 4-A audiometer consists of a spring motor phonograph using a magnetic reproducer instead of the usual acoustic reproducer. The magnetic reproducer picks up the vibrations originated by the record and transforms them into electrical vibrations. These in turn are conveyed to the telephone head set by this transformed into sound waves and delivered to the ear of the person or persons under examination who hear as if by telephone.

The records employed with the audiometer are made especially for use with this instrument. They are so arranged that the intensity of the sounds (numbers) transmitted to the listener's ear decreases in small steps to a minimum, returns abruptly to the maximum and then decreases again. This process occurs four times in the playing of each side of the double-faced record. The first two series of numbers on each face are spoken in a woman's voice and the second two in a man's. Each decreasing series is composed of different numbers since repetition would introduce the memory factor into the test. The same rate of intensity attenuation is, however, maintained in all four series. Each ear of the patient can therefore be tested four times at each intensity. The determination of hearing loss can thus be made with considerable accuracy.

For the use of those being examined special data sheets (Fig. 354) have been designed. The listener writes on these special forms the numbers heard, thus indicating the sound intensity at which intelligibility ceases. This intensity determines the person's acuity of hearing.

By placing a master sheet by the side of the data sheet so that the horizontal lines coincide, the examiner can immediately discover what figures have been recorded incorrectly and can thus determine the patient's hearing loss.

**Tuning Forks**—The Bezold Edelmann set of forks and whistles has become standard. It is constructed upon scientific principles and should be used by all otologists. It covers the range of hearing of the human ear. The forks are weighted and free from overtones. With them deaf mutes may be tested for islands of hearing and when found the islands or areas of the organ of Corti which are functioning may be utilized to teach speech if within the range of tones used in articulate

speech. They are indispensable for scientific work. No other set of forks and whistles meets all the demands. One may usually, though not always do diagnostic work with three well-selected forks, for instance the Reimer set consists of one C (64 cycles) for estimating the low tones, one  $d\sharp$  (1338 cycles) for the relative bone and air conduction, and  $c^4$  (2048 cycles) for estimating the high tones. While one may not determine the low or the high limits with these a loss of hearing for low tones or for high tones may be determined by the shortening of the time the C or  $c^4$  forks respectively are heard as compared with the normal.

**Magnesium Forks** — Magnesium forks are rustless and of light weight. They consist of magnesium 95.6 per cent manganese 0.4 per cent and aluminum 4 per cent.

So far as duration of vibration is concerned, the alloy forks seem to be as good as the steel ones excepting the  $c^5$  (4096 cycles). At some of the pitches, the magnesium forks appear even better than some of the steel ones, but on the other hand, some of the medium low-pitched steel forks vibrate a little longer. The low-pitched magnesium forks with their long prongs have rather decided overtones but these can be largely eliminated by carefully striking the fork at certain points with a soft object like a cloth hammer or the side of the hand. In order to definitely obviate the overtones weights may be used as is done with the low-pitched steel forks, or a wide rubber band may be placed about each prong as suggested by Bezold many years ago.

**Principles Underlying the Tests of Hearing**<sup>1</sup>—1 When the conduction apparatus is diseased or obstructed the hearing is impaired or lost principally for the lower tones of the scale.

2 When the perception apparatus is diseased the hearing for all tones is impaired but the loss is greater for the high tones.

3 The normal ear hears the tuning fork about two times as long by air as by bone conduction. The ratio varies with different forks.

4 When the conduction apparatus is diseased or obstructed, bone conduction is increased and air conduction is diminished. Bone conduction may be so much increased that the fork is heard longer by bone than by air conduction (Negative Rinne).

5 When the perception apparatus is diseased bone conduction is diminished. Hearing for the tuning fork by air conduction is diminished to a less degree so that it appears relatively exaggerated.

**The Voice Test** — The practical test of hearing is the ability to hear conversation, but as the spoken voice is usually too loud for the distance obtainable in an ordinary office, and as there is a great difference in the carrying quality of different consonants and vowels the whispered voice is more applicable, provided the deafness is not of such degree that the whisper is heard with great difficulty or not at all. In using the whisper it should be given with only the residual air so as to obtain the

<sup>1</sup> The following portion of the Functional Tests of Hearing was originally written by the author (W. L. B.) and revised for the fourth edition by Dr. A. I. Lewis. Subsequent revisions and additions have been made by the co-author (H. C. B.)

greatest degree of uniformity, and the examiner should train his voice to this end. In a perfectly quiet long narrow hall the whisper may be heard by a normal ear at about 40 feet. However in the average office room the whisper may not be heard more than 20 feet. In 1871 Oscar Wolf published his conclusions as to the voice as a means of testing the organ of hearing. He found the letter R the lowest in the scale while the highest number of vibrations were produced by the letter S. In the same manner some words are high pitched while others are low. Examples of high pitched words are six, seize, tease, message, shady, low-pitched words horror, rural, moon, rude, medium pitched words table Mary, baby.

To a certain degree the voice test may indicate the form of deafness as, for instance, in conduction deafness the high pitched words are heard much better than the low ones, as a rule. The reverse is not true to any degree in nerve deafness, however, in this condition the F sounds may not be heard.

*Technic* — Place the patient at one end of a quiet room with the ear to be tested toward the examiner's end of the room. The patient should not see the lips of the physician during the test. Some deaf people become very expert in lip reading.

Have the patient moisten the tip of his index finger and insert it firmly into the meatus of the ear which is not being tested. The physician should himself see that this is properly done.

The physician begins the test from without the range of the patient's hearing, approaching quietly until the patient repeats correctly what is spoken or whispered to him, and the distance so found is entered on the record. If the room is not long enough the physician should turn his back to the patient. If the distance is still too short the patient

should turn his open ear to the opposite wall. Each of these maneuvers is supposed to indicate an increased distance of about one-third. The spoken or the whispered voice is used according to the degree of deafness and the record should state which style of speech is used. Repeat test with the other ear, using different words or numbers.

Inflate the ears and repeat the test and record the difference, if any, following inflation.

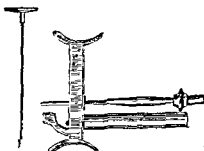


FIG. 38a — Politzer's acoumeter

**The Politzer Acoumeter** — This instrument was designed to give an accurate mechanical standard of measurement for the hearing distance for high tones. All the instruments are supposed to be of the same pitch and timbre, and the hearing distance for them in a quiet room should be about 40 feet.

**The Watch Test** — The watch has long been used to test the acuteness of hearing. As a diagnostic aid it is far inferior to other tests, but is

mentioned because of its common use. It may be used to measure the hearing distance by approaching the ear with it by firm contact with the auricle if not heard by air conduction by contact with the mastoid process, by placing it between the teeth and noting in which ear it can be heard most plainly, as in the Weber experiment, and for comparison from time to time during treatment. Its drawbacks are: Watchticks are not standardized and is a test for those frequencies only that the watch may produce, the hearing for the watch is no indication whatever of the hearing for conversation and the patient becomes accustomed to the sound of one watch and apparently hears it better when there is no real improvement in hearing.

**The Weber Test**—E. H. Weber first found that a normally vibrating tuning fork (Fig. 386) placed upon the skull is much more distinctly

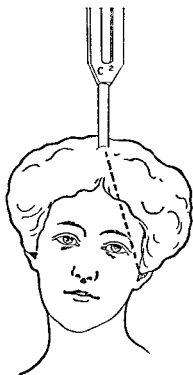


FIG. 386.—The Weber test. The patient is deaf in the left ear and the sound lateralizes to the left ear, thus indicating disease of the sound-conduction (middle ear) apparatus of the left ear.

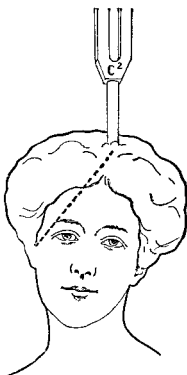


FIG. 387.—The Weber test. The patient is deaf in the left ear and the sound lateralizes to the right or good ear, thus indicating disease of the perception apparatus (internal ear or eighth nerve) of the left ear.

heard in the ear which is closed by the finger inserted in the meatus. In other words, the sound is referred to that ear in which a conduction deafness has been produced. Clinically it has been shown that when the middle ear alone (including the eustachian tube) is diseased, or when the external canal is obstructed, the sound of the vibrating tuning fork when on the median line of the skull as the vertex, forehead, teeth

or chin is lateralized toward the affected ear and that when the internal ear alone is affected the sound is lateralized toward the unaffected ear. This test is not altogether dependable in bilateral deafness nor in unilateral deafness when both the middle and internal ear are affected as there are two opposing conditions one tending to increase while the other tends to decrease bone conduction. Often also patients do not detect the lateralization of the sound or are inaccurate. Hence this test should be accepted only in corroboration of the other tests if inconsistent with them it should be ignored. In suppurative disease of the ear if the sound is first referred to the infected ear but subsequently changes and is referred to the well ear it is very suggestive of involvement of the labyrinth.

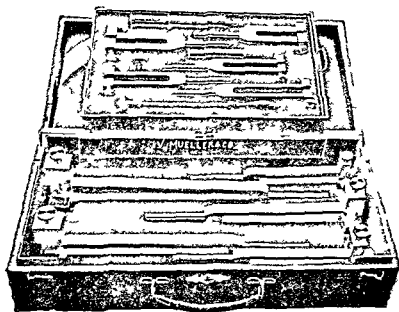


FIG. 388.—Bezold Edelmann's tuning forks and whistles

*Technic*—The Bezold large *A* fork (1087 cycles) or any fork between this and *c* (512 cycles) may be used. The *c* is commonly used but is rather high. The vibrating fork is placed in the median line on either the vertex forehead glabella teeth or chin and the patient asked to indicate in which ear the sound appears to be loudest. This is entered on the record. Patients often have the preconceived notion that they must hear it louder in the better hearing ear. This should be overcome. In order to test the accuracy of the answer the following simple procedure will often suffice. If the patient says, "I hear it louder in my right ear," the examiner closes the right ear with his finger (the fork meanwhile remaining in place and vibrating) and asks, "Now where do you hear it?" If the patient then imagines the sound is referred to the open ear he may be known as inaccurate.



**The Schwabach Test**—Schwabach first noticed that the sound of the tuning fork through the cranial bones in conduction deafness was heard longer than normal. The explanation of this is still open for discussion. It is at least partially due to the interference with the entrance of adventitious sounds from without, and with corresponding interference with the egress of some of those received through the cranial bones. The practical application of this is the comparison of the bone conduction for the tuning fork with the known normal for that fork. This test is very valuable in connection with the relative air and bone conduction test (Rinne test). Markedly prolonged bone conduction indicates a conduction deafness. Markedly shortened bone conduction indicates an internal ear lesion or disturbance, *i.e.*, nerve deafness. This is not necessarily an organic lesion (Lewy<sup>1</sup>). Sometimes when the patient ceases to hear the fork by bone conduction if the fork is removed for a few seconds and then replaced without having been struck again the patient again hears it. This is known as the 'fatigue symptom', and is supposed to be due to fatigue or exhaustion of the nerve. In combined cases *i.e.*, cases of mixed conduction and perception deafness, the Schwabach test may show the bone conduction somewhat shortened, slightly lengthened or approximately normal. On account of the affection of the perception apparatus the disturbance of the conduction apparatus fails to bring about the usual increase in bone conduction.

**Technic**—The fork for this purpose should be free from overtones, not so low that the vibrations are transmitted as concussions to the skull nor so high that it is difficult to distinguish between the air and bone conduction. The best fork is the Bezold-Edelmann *A* (108.7 cycles) as recommended for the Weber test. Any good fork of sufficient intensity and duration, free from overtones between *A* and *c*<sup>2</sup> may be used, but the Reimer *d*<sub>4</sub><sup>2</sup> (153.8 cycles with clamps) is next choice. The normal register of the fork must have been ascertained previously by trials on normal persons. In order to reproduce even results the fork must always be struck on the same object in the same manner and with the same force, and applied to the skull of the patient with the same degree of pressure. For instance, in using the Bezold *A*, the fork may be dropped from the vertical to the horizontal by its own weight, striking on the examiner's knee (the examiner's thigh is flexed to a right angle with his body and the handle of the fork just touches the thigh at the beginning of the fall), the fork is then rested by its own weight on the patient's skull. Or one may use a small rubber hammer made for the purpose of striking the tuning fork, or the rubber hammer used by neurologists for obtaining tendon reflexes will do. The number of seconds from the stroke until the patient no longer hears the fork is noted, preferably on a stop-watch and entered on the record "Schwabach—seconds". (If preferred one may record the per cent of normal, *e.g.*, *S* 40 per cent or 150 per cent.) The patient must be instructed to raise

<sup>1</sup> *Laryngoscope*, (March) 1913

his hand or otherwise indicate the moment he no longer *hears* (not feels) the fork.

**The Rinne Test** (Combined Testing of Bone and Air Conduction) — This is a very valuable test. If one holds the handle end of a vibrating tuning fork against the mastoid process until the tone is no longer heard, and then brings the prongs near the external auditory meatus (Figs 389 and 390) the sound will again be heard, the length of time the tone is heard through the air being double or treble, according to the fork used the hearing time through the bone. This is the "Positive Rinne." It occurs normally. It also occurs in nerve deafness though

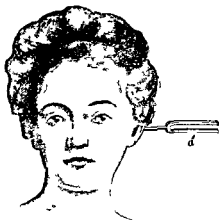


FIG 389 — Showing the Pinna *a* (435 cycles) fork in position on the mastoid process in the Rinne test

FIG 390 — Showing the Rinne *a* (435 cycles) fork held close to the ear in Rinne's test indeed the prong tips should be within the concha

in this condition both the bone and air conduction are shortened — "Shortened Positive Rinne." In a pure conduction deafness the bone conduction is relatively lengthened while the air conduction is relatively shortened. When this condition advances to a point where bone conduction exceeds air conduction we have a "Negative Rinne." Negative Rinne, but with both bone and air conduction very much shortened, may also occur in severe nerve deafness. Plus-minus Rinne is a term applied when bone and air conduction are equal. Indefinite Rinne when air conduction is entirely absent. False Rinne, when one ear is totally deaf and the fork apparently heard on the mastoid of the deaf ear is really heard in the other ear. The bone conduction as found with the Rinne test should corroborate the Schwabach test. Occasionally bone conduction is prolonged for the *A* fork, and shortened for the *a*. This condition has been found in syphilis in a few cases.

**Technic** — The best fork for this purpose is the Edelmann *a* (435 cycles). It is free from overtones and of sufficient intensity and duration, and yet the tone is not carried through air conduction to the opposite ear from the one being tested, *c* (128 cycles), *d* (153.8 cycles), *e* (256 cycles) and *e* (512 cycles) may also be used if they fulfil the above

indications The fork for this test should be carefully selected as it is the most frequently useful one The same degree of force and the same object (non metal) for striking should always be used Striking the fork on the knee does very well It is then firmly placed with the end of the handle on the mastoid process over the antrum being held by the handle near the prongs Care should be observed to use uniform pressure and to avoid contact with the auricle or hair When the patient indicates that the sound is no longer heard the fork is held suspended with the prongs flatwise toward and as near as possible to the concha with out touching In this position the sound is heard best and longest If abundant hair prevents the fork being held in this manner it may be held prongs up If the Schwabach test shows greatly increased bone conduction it often saves time to get the air conduction first in making the Rinne test as it will probably be a negative The length of time the fork is heard by bone conduction and by air conduction is measured in seconds preferably with a stop watch from the time the fork is struck one stroke sufficing for both parts of the test and is entered on the record Rinne + 12 30 or Rinne - 20 15 for example the bone conduction first or if written as a fraction ( $\frac{12}{30}$ ) the bone conduction is the numerator The normal register for the fork used must be known

**The Gellé Test** If the air is compressed in the external canal of the normal ear (using an air bag with a snug fitting ear piece) the tone of a vibrating tuning fork placed on the vertex or mastoid will be perceived greatly diminished According to Gelle if the stapes is anklylosed the pressure in the external canal cannot be transmitted to the labyrinthine fluid and the test is then negative A more practicable method of performing this test as devised by Barany is as follows A branched or T-shaped auscultation tube is used two ends carrying snug fitting ear pieces the third a mouth piece One of the earpieces is held tightly by the patient in his external meatus so that no air escapes the other likewise by the examining physician the third is used to compress the air in the tube and in the external canals by the physician blowing into it The stem of the vibrating tuning fork is placed about the middle of the rubber tubing If the stapes is not anklylosed both the patient and the physician will hear the sound greatly diminished during compression of the air in the tube (unless the hearing is already very poor) Thus the examiner has a control test

**Bing Test — No 1** — The small end of a speaking trumpet is fitted into the free end of a catheter which is inserted into the eustachian tube so that the sound waves enter the cavum tympani and come into direct contact with the foot plate of the stapes and the membrane of the round window If the speech is heard decidedly better in this way than with the end of the speaking trumpet in the external meatus the inference is that the interference with conduction is outside the stapes that is in the incus malleus or drum membrane

**No 2** — Bing claims that after the tone of a vibrating tuning fork on the vertex becomes inaudible it is again heard if the meatus is occluded with the finger if there is a labyrinthine affection As this occurs normally, the test is useful only in severe deafness If there is a con

his hand or otherwise indicate the moment he no longer *hears* (not feels) the fork.

**The Rinne Test** (Combined Testing of Bone and Air Conduction) — This is a very valuable test. If one holds the handle end of a vibrating tuning fork against the mastoid process until the tone is no longer heard and then brings the prongs near the external auditory meatus (Figs 389 and 390) the sound will again be heard. The length of time the tone is heard through the air being double or treble according to the fork used. The hearing time through the bone. This is the *Positive Rinne*. It occurs normally. It also occurs in nerve deafness though

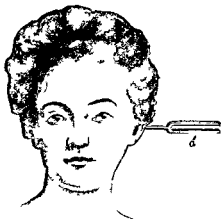


FIG 389 — Showing the Positive (435 cycles) fork in position on the mastoid process in the Rinne test



FIG 390 — Showing the Rinne a (435 cycles) fork held close to the ear in Rinne test indeed the prongs should be within the concha

in this condition both the bone and air conduction are shortened — **Shortened Positive Rinne**. In a pure conduction deafness the bone conduction is relatively lengthened while the air conduction is relatively shortened. When this condition advances to a point where bone conduction exceeds air conduction we have a **Negative Rinne**. **Negative Rinne** but with both bone and air conduction very much shortened may also occur in severe nerve deafness. **Plus-minus Rinne** is a term applied when bone and air conduction are equal. **Indefinite Rinne** when air conduction is entirely absent. **False Rinne** when one ear is totally deaf and the fork apparently heard on the mastoid of the deaf ear is really heard in the other ear. The bone conduction as found with the Rinne test should corroborate the Schwabach test. Occasionally bone conduction is prolonged for the *A* fork and shortened for the *a*. This condition has been found in syphilis in a few cases.

**Technic** — The best fork for this purpose is the Edelmann *a*<sup>1</sup> (435 cycles). It is free from overtones and of sufficient intensity and duration and yet the tone is not carried through air conduction to the opposite ear from the one being tested. *c* (128 cycles) *d*<sub>7</sub> (153.8 cycles) *c*<sup>1</sup> (256 cycles) and *c*<sup>2</sup> (512 cycles) may also be used if they fulfil the above

indications The fork for this test should be carefully selected, as it is the most frequently useful one The same degree of force and the same object (non-metal) for striking should always be used Striking the fork on the knee does very well It is then firmly placed with the end of the handle on the mastoid process over the antrum, being held by the handle near the prongs Care should be observed to use uniform pressure and to avoid contact with the auricle or hair When the patient indicates that the sound is no longer heard, the fork is held suspended with the prongs flatwise toward and as near as possible to the concha without touching In this position the sound is heard best and longest If abundant hair prevents the fork being held in this manner it may be held prongs up If the Schwabach test shows greatly increased bone conduction it often saves time to get the air conduction first in making the Rinne test, as it will probably be a "negative" The length of time the fork is heard by bone conduction and by air conduction is measured in seconds, preferably with a stop-watch, from the time the fork is struck, one stroke sufficing for both parts of the test and is entered on the record "Rinne + 12 35" or "Rinne - 20 15" for example, the bone conduction first, or if written as a fraction ( $\frac{35}{20}$ ) the bone conduction is the numerator The normal register for the fork used must be known

**The Gellé Test**—If the air is compressed in the external canal of the normal ear (using an air bag with a snug fitting ear piece) the tone of a vibrating tuning fork placed on the vertex or mastoid will be perceived greatly diminished According to Gellé if the stapes is ankylosed the pressure in the external canal cannot be transmitted to the labyrinthine fluid and the test is then negative A more practicable method of performing this test as devised by Barány, is as follows A branched or 'T'-shaped auscultation tube is used, two ends carrying snug fitting ear-pieces, the third a mouth piece One of the earpieces is held tightly by the patient in his external meatus, so that no air escapes, the other likewise by the examining physician, the third is used to compress the air in the tube and in the external canals by the physician blowing into it The stem of the vibrating tuning fork is placed about the middle of the rubber tubing If the stapes is not ankylosed, both the patient and the physician will hear the sound greatly diminished during compression of the air in the tube (unless the hearing is already very poor) Thus the examiner has a control test

**Bing Test**—No 1—The small end of a speaking trumpet is fitted into the free end of a catheter, which is inserted into the eustachian tube so that the sound waves enter the cavum tympani and come into direct contact with the foot plate of the stapes and the membrane of the round window If the speech is heard decidedly better in this way than with the end of the speaking trumpet in the external meatus, the inference is that the interference with conduction is outside the stapes that is, in the malleus or drum membrane

No 2—Bing claims that after the tone of a vibrating tuning fork on the vertex becomes inaudible it is again heard if the meatus is occluded with the finger, if there is a labyrinthine affection As this occurs normally, the test is useful only in severe deafness If there is a con-

duction deafness the sound is not again heard when the meatus is occluded

**The Low Limit**—Normally the low limit is about  $C^2$  (16 cycles) but some persons with otherwise normal hearing fail to distinguish this tone. However failure to hear  $G$  (24 cycles) may be interpreted as indicating some loss of hearing for lower tones. Bezold states that failure to hear  $C^1$  (32 cycles) in conduction deafness indicates ankylosis of the stapes. Loss of hearing for low tones practically always occurs to some degree in conduction deafness the greater the loss the greater the probability of stapes ankylosis. It rarely occurs in pure nerve deafness (except congenital) but of course occurs in combined cases.

**Technic**—The examiner begins with the lowest fork. If this is not heard the next one higher is used first before one ear then the other until the patient whose eyes should be closed during the procedure indicates that he hears the tone. The lowest fork heard by each ear is entered on the record. If one uses but one fork for estimating the hearing for low tones  $C$  (64 cycles) is a practicable one. It must be weighted to prevent overtones and its normal register must be known. One can then enter on the record the fraction or percentage of time as compared with the normal that this fork is heard. Shortening of the hearing time indicates loss of hearing for low tones.



FIG. 391 Test of the hearing with the Galton Edelmann whistle at 18 inches

**The High Limit**—Edelmann states that the educated ear can distinguish  $f^1$  (22 097 cycles) on a good Galton whistle and  $d^1$  (18 581 cycles) on a Schulze or the Struven-Schaefer monochord. However many normal ears do not hear beyond  $c^1$  (16 384 cycles). After the age of about fifty years the high limit declines owing to senile changes. A slight decline may begin as early as twenty five years of age. According to Zwaardemaker at the beginning of senility the limit is about  $a^6$  in old age about  $g^6$ . These data should be borne in mind in estimating the significance of tests for the range of hearing. Any marked loss of the upper range indicates some pathologic process in the internal ear. When associated with a conduction deafness it indicates the probability of beginning degeneration in the basal turn of the cochlea. A moderate loss for high tones occurs quite commonly in otosclerosis of the labyrinthine capsule.

*Technic*—The *Galton whistle* (Edelmann's) has an adjustable aperture and graduated pipe-length, both operated by screws, and is blown by compression of a bulb. A scale for translating the tones into their proper musical designation accompanies each instrument. The whistle must be blown gently as it is difficult to exclude the opposite ear even when the meatus is occluded. Begin above the high limit and gradually lengthen the pipe by the screw until the sound is heard as a clear whistle (as distinguished from the blowing sound). The number of the line and the aperture distance or its equivalent in musical terms is entered on the record. The small whistles are practically useless except in cases with very marked loss of upper tone limit. All whistles of course test only air conduction.

The *Monochord* consists of a metal frame on which is stretched a piano wire. On the frame and wire is fitted a block which by its position

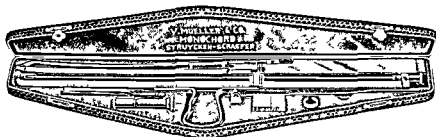


FIG. 392.—Monochord

regulates the pitch. A bone button can be attached to one end of the frame, which is held in contact with the mastoid process to test bone conduction for high tones, an advantage which this instrument has over the whistle. Transverse vibrations are caused by striking the wire with a small hammer or drawing a violin bow across it. For the highest tones longitudinal vibrations are used and these are obtained by rubbing the wire lengthwise with a felt pad moistened with turpentine and benzole, or carbon tetrachloride. (A felt-tipped bottle which keeps automatically moist is furnished with the instrument.) The range of the Struycken-Schaefer monochord is from  $g^1$  to above the high limit. The frame is calibrated so that the pitch can be read directly for longitudinal vibrations, and in centimeters for the transverse vibrations, which requires reference to a scale for translation into musical terms. In obtaining the transverse vibrations (low tones) the instrument must be rested on a table to act as resonator, as otherwise the tone is too thin. In obtaining the high tones the patient must distinguish between the rubbing and the clear tone but this is not difficult.

The  $c^4$  (2048 cycles) fork (large size) of either the Bezold, Hartmann or the Reimer set may be used to test the high limit quite satisfactorily and more simply than above described. The fork is stroked gently, tapped with the finger or struck on metal according to the degree of loss for the upper tones, or the examiner can by alternately holding the vibrating fork before the patient's and his own ear (if normal) determine if the upper limit is normal, slightly short, moderately short or very

much short and so enter it on the record. The small size  $c^4$  forks seldom vibrate long enough to perform this test satisfactorily.

Sonnenschein points out the common errors in making the functional fork tests as due to (1) lack of knowledge of the pitch of the fork used in making the tests (2) lack of knowledge of the length of time the tuning fork vibrates audibly for the normal hearing ear both by air and by bone conduction (3) lack of attention to the position in which the fork is held while the air conduction test is being made (4) lack of attention to the normal fatigue of the end-organ (5) indifference to the manner in which the fork is applied to the mastoid when the bone conduction test is being made (6) lack of knowledge as to whether the control hears normally or not and (7) lack of attention to the age of the control as compared with that of the person being examined.

**Unilateral Total Deafness**—If both ears are occluded by the moistened fingers a loud fork or voice can still be heard. It is evident that though one ear be totally deaf the other cannot be entirely excluded from hearing by simple occlusion of the meatus; therefore it is necessary to use one of several methods that have been devised, all of which operate both by occluding and producing noise in the ear which is to be put out of commission temporarily while the supposedly totally deaf ear is tested. When one of these devices is properly applied to one ear, if the other is totally deaf the patient will not hear even a loud voice (unless shouted directly toward the head).

**Wagner's Shaking Test**—A moistened or dry finger is tightly inserted in the external auditory meatus. With the patient's eyes closed or blindfolded a shaking movement of the inserted finger is produced resulting in a complete occlusion for the whisper and moderately loud conversational voice.

**The Neumann Noise Apparatus**—This is an electrical device which operates with either a direct or an alternating current. It consists of a rheostat and interrupter and two telephones fitted with ear-pieces which fit snugly into the external meatus. A switch causes the noise to be heard in either or both ears at the will of the operator. The rheostat controls the intensity of the noise. The instrument can also be used to discover malingerers who claim unilateral deafness.

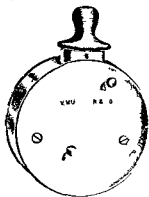


FIG. 393. Bárány's noise apparatus.

**The Barany Noise Apparatus**—This is a clockwork buzzer which when wound up is operated by pressing a button while the ear piece is in place. It is probable that sounds above  $c^2$  (512 cycles) are not completely excluded by this or the Neumann noise apparatus or Wagner's shaking test.

**The Pierce Method**—A C fork (64 cycles) of sufficient loudness and duration (the one recommended for the low limit or a Koenig C fork will do) is fitted on the handle end with a conical ear piece. The ear



piece is moistened and while the fork is vibrating loudly it is placed firmly in the external meatus

Other methods are the running of a stream of water or air under slight pressure into the canal of the ear to be excluded

**Tests for Hearing in Infants**—In very young infants all tests for hearing have been dependent on visible response of the baby to sounds. This response in the baby has presumably been dependent on the inherent fear of sudden or unusual sounds noted in practically all infants. This reaction usually consists in a sudden start of the entire body a winking of the eye a crying out or a combination of these activities

The vestibular tests both turning and caloric may be given. If normal or subnormal vestibular reactions are found it may be assumed that some degree of cochlear function is present. If however the vestibular tests give no response and there is no other evidence of hearing the deafness may be regarded as profound and with a very poor prognosis

**Conditioned Reflex Test**—C. A. Aldrich<sup>1</sup> suggests a test for hearing in the new born based on a conditioned reflex with sound as one stimulus and pain as the other. He reports his case as follows

At half hour intervals during the night a small dinner bell was rung beside the baby's crib in such a way that she could not see it while at the same time the sole of the right foot was firmly scratched with a pin. When these stimuli were applied the child cried out lustily and drew up the right leg. By mid morning after perhaps twelve or fifteen applications the infant cried and drew up the leg when the bell was rung and *the foot was not touched*. No one could see the experiment without being perfectly sure that the child heard. If the bell was slightly tinkled her face at once looked worried. The worried look was followed by a cry when the bell was loudly shaken.

## SIMULATED DEAFNESS

Various motives lead to simulation of ear disease. Hysterical individuals sometimes do it to excite attention or sympathy. Soldiers in the army and men drafted to fill the ranks who desire to avoid duty and those injured on railways streets and in shops who wish to collect damages through the courts sometimes exaggerate or assume deafness or artificially produce ear disease.

**Tests for Simulated Deafness**—First make a careful examination of the external ear auditory canal drumhead and the eustachian tube. Most cases of suspected simulated deafness are unilateral. This arises from the fact that a bilateral deafness would have previously attracted attention whereas a one-sided deafness might have existed without being discovered. In other words it is easier to simulate one-sided deafness hence its greater frequency among malingerers. They often artificially produce an obvious cause for the deafness they wish to assume by dropping strong solutions of silver nitrate carbolic acid

creosote tincture of cantharides etc into the auditory canal. The skin and drumhead are thus cauterized and simulate in some degree suppurative otitis media. A careful examination will usually reveal the source of the inflammation. If silver is used a dark brown stain will be seen whereas if carbolic acid is used the bleached skin will aid in arriving at a correct conclusion. A bandage placed over the ear and sealed will in these cases lead to a speedy recovery as the malingerer is unable to continue the caustic applications. Foreign bodies placed in the canal to simulate deafness and ear disease may be detected by a careful examination.

It is in cases in which there are no objective signs of ear disease that the real difficulty of detecting malingering arises. The would be patient often studies the subjective signs of labyrinthine deafness so well that if he is especially shrewd it is difficult to detect him. In making the examination of this class of cases the eyes of the suspect must be bandaged thus rendering it somewhat difficult for him to judge distances in testing with the voice fork or watch. If he hears the instrument at greatly varying distances with the deaf ear (the other being tightly plugged) it is fair to presume he is malingering. If on the other hand during repeated short testings he hears at about the same distance it is fair to presume that he is really deaf.

Since the advent of the audiometer detecting the malingerer is somewhat easier as it is almost impossible to produce two or more audiograms of simulated deafness with enough accuracy to be convincing.

**Erhard's Test**—When a normal ear is tightly closed a loud ticking watch may be heard at 3 or 4 feet. The patient should have the supposedly deaf ear tightly closed and when the watch is within 3 or 4 feet of the normal ear he should be commanded to count the beats which he will of course readily do. The sound ear should then be closed the supposedly deaf one being open and the same test made on the open deaf ear. If when the watch is within 2 or 3 feet of the ear he says he does not hear it it is fair to presume that he is simulating the deafness as at that distance he would hear the watch with the closed normal ear.

**Chimani Moos Test**—In one-sided deafness a large vibrating c-fork is alternately held at an equal distance from each ear until the suspected malingerer makes it plain to himself that he hears the fork loudest before the normal ear. The vibrating fork is then placed on the vertex bridge of the nose or median line of the incisor teeth and the patient is asked in which ear he hears the fork the plainer. A patient with true unilateral middle-ear disease will without hesitation say that he hears it louder on the affected side whereas a malingerer will hesitate as he hears it equally well on both sides or he may say he hears it only in the good ear. The normal ear should now be tightly closed and the vibrating fork again placed on the median line of the skull and the malingerer will probably say he does not hear it at all or but faintly whereas the sound should be heard more distinctly in the good ear with the auditory canal closed.

**Stethoscope Test.**—A common stethoscope, having one tube closed with a wooden plug, may be used to detect simulated unilateral deafness. The stethoscope should be adjusted to the patient's ears, the open tube leading to the suspected ear, the closed one to the normal ear. The physician should now speak into the bell of the stethoscope, having the patient repeat what he hears. The instrument should then be removed, the normal ear tightly closed, and the same formula repeated to the patient. He will say he cannot hear, whereas he has already repeated after you, with the normal ear tightly closed with the plugged arm of the stethoscope. In other words, he heard with his suspected ear through the open tube of the stethoscope (the one leading to the normal ear being tightly closed), thinking, of course, that he would lead the examiner to believe he heard with the normal ear.

**Ear Specula Test.**—The use of four ear specula, two open and two half filled with wax, may be used to detect malingering. The patient should sit with bandaged eyes facing the wall. The two open specula should be simultaneously introduced, one in each ear, and the examiner (behind the patient) should repeat certain words, or numerals, at varying distances, and thus ascertain his hearing distance with both ears open. He should then change the specula, using one open and one closed, then two open, then two closed, and so on, noting the distances he hears with the varying combinations of the specula. In this way the patient will unwittingly reveal the true condition of his hearing apparatus.

Repeated examinations and the striking contradictions made by the malingerer during the various examinations will lead to a correct diagnosis in most cases.

**Lombard's Test.**—Bárány's noise apparatus may be used to detect malingering in one-sided deafness. The patient reads some selected paragraph or article aloud. So long as he hears his own voice it does not change in pitch or articulation. The noise apparatus is then applied to his sound ear while he continues reading. If he is actually deaf in the so-called affected ear his voice will become elevated in pitch and the articulation blurred. If he hears with that ear his voice will remain unaffected. This test may be made experimentally upon normal individuals by using two Bárány apparatuses. At the beginning of the reading one is applied to the right ear. After a few sentences are read the other is applied to the left ear, thus rendering the patient totally deaf. His voice and articulation will be greatly modified.

**Stenger's Test.**—This test is used for the detection of a simulated total unilateral deafness. It is based on the fact that a preponderance of sound of a certain pitch in one ear eliminates the perception for sound of the same pitch in the opposite ear. The test is made as follows:

The patient is blindfolded. Two forks of the same pitch and intensity are selected. The hearing distance of the good ear for one of the forks is carefully determined. The fork is then struck and held 1 inch from the bad ear. The patient will probably deny hearing it. If he does so the second fork is struck and slowly brought toward the good ear.

With equal hearing in both ears and with both forks vibrating with the same intensity the patient will not hear the fork in the good ear until it is in the same relative position (1 inch) as the fork held before the allegedly bad ear. If the patient is really deaf in one ear the fork should be heard at a greater distance in the good ear.

**Teals Test**—This test is used for the detection of a simulated unilateral deafness. The usual tuning fork tests are made in which he denies hearing by air conduction in the allegedly deaf ear. He is then blindfolded and the bone conduction over the mastoid on the deaf side is tested. He usually admits hearing it. If he does not admit hearing it his answer is open to question as with one good ear the sound should be carried through the bone. The real test is now used. He is told the last test is to be repeated. A non-vibrating fork or the end of a pen or pencil is placed against the mastoid of the deaf ear and a vibrating fork (the same fork formerly used in testing the air conduction) is held a short distance from the auricle of the same ear. If he is really deaf he of course will not hear the fork. If he hears it he is discovered in his simulation as it must have been by air conduction.

**Callahan's Test**—In this test for unilateral simulated deafness two strands of rubber tubing with a central mouthpiece are used. Couplings of additional rubber tubing are provided. The test is based on the fact that with unequal lengths of the tubing the patient is conscious of hearing the voice in the ear connected with the short length tubing. The patient will admit hearing in the good ear (within the hearing limits of that ear). However, when he perceives the voice in the supposedly deaf ear he will deny hearing anything and as a result is detected.

**Bilateral Simulated Deafness**—This is much more difficult to detect. Constant observation or catching the patient off his guard will usually unmask the simulation.

A modification of the Lombard test for bilateral simulated deafness consists of sounding a Barany noise apparatus in both ears while the patient is reading; if he elevates his voice he is simulating.

Repeated tests with an audiometer for partial bilateral deafness will produce great variations in the graph due to the patient's inability to remember the intensity of the various frequencies to which he claims to be deaf.

**Cochleo-palpebral (Gault) Test**—The cochleo-palpebral test of Gault is of value in bilateral as well as unilateral deafness. It is made by occluding the good ear and then producing an unexpected loud noise near the deaf ear. If a slight contraction of the lid occurs hearing is indicated.

**Caloric Test**—If the caloric tests show an absent or depressed vestibular reaction impairment of hearing may be suspected also.

## CHAPTER XXXIX

### MALFORMATIONS AND NEOPLASMS OF THE EAR

#### MALFORMATIONS OF THE AURICLE

MALFORMATIONS of the auricle are of importance chiefly from a cosmetic point of view. The auricle plays such a small part in the function of audition that its entire absence does not materially influence the acuity of hearing. If however the auricle is so shaped as to occlude the meatus it may interfere to some extent with the transmission of sound waves and thus impair hearing. In most cases however when there is a very marked defect there is also defective formation of the external auditory canal and the middle ear due to the common branchiogenic development of these parts. The labyrinth is normal as a rule.

The malformations may be of a great variety of forms ranging from a plurality of the auricle to its entire absence. Between these two extremes the auricle may be deformed to a slight degree or it may be overdeveloped or misshapen in almost every conceivable way. It may be either arrested (microtia) or overdeveloped (macrotia). One part may be overdeveloped while in another the development is arrested.

The defect may be either congenital or acquired. If congenital it is due to a maldevelopment of the first and second visceral arches and the first visceral or pharyngeal cleft from which the external and middle ears develop. It may be unilateral or bilateral usually the former.

Auricular appendages or supernumerary auricles consist of reticular cartilage subcutaneous cellular tissue and skin. They are usually located in front of the tragus although they may be on the lobule the side of the neck or the shoulders.

The acquired form of malformations of the auricle may be due to injuries hematomas abscesses infections chondritis perichondritis etc.

**Macrotia**—Macrotia or lop ear may be corrected surgically as follows. The skin on the posterior surface of each auricle is incised with a knife. The line of incision extends in a curve from within  $\frac{1}{4}$  inch of the superior attachment of the auricle to within  $\frac{1}{2}$  inch of its inferior attachment. A second incision is begun at the upper point and extended backward and downward over the mastoid process  $\frac{1}{2}$  inch posterior to the attachment of the auricle and made to join the inferior end of the auricular incision (Fig 396). An ellipse or segment of skin not unlike a segment of orange peel is thus outlined. This is dissected from the auricle and the mastoid process.

The second step of the operation consists in cutting through the cartilage of the auricle following the line of the auricular skin incision. The cartilage is then severed at the auriculo-mastoid junction care being exercised to avoid cutting through the skin on the anterior surface.

of the auricle. The cartilage is next carefully separated from the anterior skin of the auricle.

The third step of the operation consists in closing the wound. This is done in such a way as to bring the auricle close to the head as the operation is done principally for this purpose. In order to do this four deep stitches are taken so as to include the auricular skin, the auricular



FIG 394

FIG 394 — Macrotia with absence of external auditory canal. Partial development of the auricle.



FIG 395

FIG 395 — Macrotia with absence of the external auditory canal. Rudimentary development of the auricle.



FIG 396 — Operation for macrotia or lop-ear. An elliptical piece of skin (a b) has been removed from the posterior wall of the auricle and mastoid process. a the area of cartilage to be removed from the concha of the auricle.



FIG 397 — The sutured incision at the close of the operation. C the cartilage removed from the concha of the auricle. D the skin removed from the posterior aspect of the auricle and the mastoid process.

cartilage, the fibrous tissue over the mastoid, and the mastoid skin. These are drawn firmly together and secured. A continuous horsehair suture may then be used to bring the edges of the skin together.

The superficial sutures are removed on the sixth day and the deep stitches on the ninth day

An antihelix can be made by an elliptic resection over the area corresponding to a normal antihelix, carried to the perichondrium, but without removing the cartilage

**Preauricular Cyst, Congenital Aural Fistula**—Preauricular cyst or congenital aural fistula, first described by Hef§inger<sup>1</sup> in 1864, is a small sinus of the external ear, which opens most commonly at the anterior border of the ascending limb of the helix. The cyst or fistula is thought to be a remnant of the first branchial cleft or faulty development of the six primitive tubercles which form the pinna. At times the condition is hereditary. The fistula opens in front of the ear either above or below the tragus, and is a blind canal filled with creamy secretion mixed with pus. When its mouth becomes closed the secretion accumulates within the canal, which may be felt as hard nodules beneath the skin. It may give rise to an offensive discharge if secondarily infected. A single retention cyst may form. It occurs more frequently in colored than in white children.

**Treatment**—The treatment consists in the excision of the tract or cyst with the complete removal of the epithelial lining. A preliminary injection of a dye into the fistula will aid in outlining the tissue to be resected.

Mild caustic applications have been applied within the canal to excite inflammation and adhesions for the purpose of closing the canal with little success.

Havens<sup>2</sup> advocates the destruction of branching or multiple tracts types of fistulas by surgical diathermy. After filling the tract with methylene blue the electric scalpel (radio knife) lays open the sinus and the epithelial lining is destroyed by electrocoagulation. The wound heals by granulation.

## HEMATOMA (OTHEMATOMA) OF THE AURICLE

**Definition**—This is a disease of the auricle characterized by an effusion of blood between the perichondrium and the cartilage.

**Etiology**—It may occur spontaneously or from direct violence. When it occurs spontaneously it is probably due to degenerative changes in the blood-vessels of the fibrous bands which traverse the cartilage of the auricle. It is also probable that degenerative changes occur in the fibrous tissue. High blood pressure may be a factor in some cases.

It is commonly found in the insane, pugilists and wrestlers. It may follow injury from any cause.

**Symptoms**—The tumor forms quickly. This distinguishes it from perichondritis, angioma or other neoplasms. It is bluish in color and is rounded and soft to the touch. It does not have the distinct fluctua-

<sup>1</sup> Arch f path Anat 29 358 1864

<sup>2</sup> Arch Otolaryngol 29 965 (June) 1939

tion common to fluid sacs beneath the skin but offers a doughy resistance. If it is due to traumatism it is usually quite large and often involves the whole or the upper portion of the auricle whereas if it is idiopathic (bleeding diseases) it is often quite circumscribed being limited to a nodule in the concha or other depression of the auricle. It is most common on the anterior or concave surface of the auricle.

Pain may be present in the traumatic variety but is absent in the idiopathic. The tumor is opaque by transmitted light whereas that of perichondritis is transparent. If the auditory meatus is occluded by the swelling partial deafness and tinnitus are present. It should be borne in mind that the deafness may be due to the rupture of the eardrum from concussion. The hematoma may become organized and cause permanent deformity.

**Diagnosis** —The diagnosis is based upon the rapid development of the growth after an injury, the opaqueness by transmitted light and the absence of febrile symptoms. In the spontaneous variety the rapid development of the tumor is quite characteristic.

**Prognosis** —The traumatic variety ends by resolution more readily than the idiopathic variety except when there is extensive damage to the cartilage. If there are no reactive symptoms and the swelling diminishes in size the prognosis is favorable. Violent inflammatory symptoms on the other hand necessitate opening the tumor thus rendering the prognosis more unfavorable. In some cases there is recovery without visible deformity while in others recovery occurs with great shrinkage or deformity of the cartilage.

**Treatment** —The treatment should be symptomatic and modified to correspond with the peculiar pathology of the case. If for example the hematoma is due to degenerative changes in the blood vessels and the connective tissue or the cartilage of the auricle it would be wrong to apply massage to promote absorption as such manipulation would probably provoke more hemorrhage. Such a procedure if tried at all should be deferred until regeneration has closed the interior wounds. Pressure bandages are also contraindicated for the same reason. The application of ice may exert a favorable influence in preventing passive inflammatory swelling and if it is already present the cold reduces it somewhat. The application of heat in the spontaneous type would seem to be better treatment as it promotes regeneration. The inflammatory type should be incised and a sterile dressing applied.

Puncture of the tumor may be done in the early stage of its development. If this is not followed by relief it is better to open it thoroughly by a free incision after which the contents are removed and the cavity packed with iodoform gauze. If the cavity is sterile apposition of the parts may be obtained by moulding dental compound or plaster of Paris in the desired shape and holding in place by means of a bandage.

Howard<sup>1</sup> recommends the window operation for hematoma of the auricle. The method consists in removing a portion of the perichondrium

<sup>1</sup>Laryngoscope 45:81 (February) 1935



and a full thickness of skin by means of a punch or other suitable cutting instruments producing a window for drainage of the fluid formations between the perichondrium and cartilage of the auricle

### CALCIFICATION AND OSSIFICATION OF THE AURICLE

**Etiology** — Calcification or ossification of the auricles is rare. Scherrer in a review of this subject found 40 cases which have been reported by 19 observers during the past sixty five years.

The age incidence varied from twenty two to eighty nine years with the majority of the cases more than fifty three years of age. The cause has been attributed to numerous factors namely unusual spontaneous changes occurring in senility with poor circulation of the peripheral parts of the body, freezing and frost bite, general debilitating diseases, trauma and perichondritis, syphilitic perichondritis or perivascular infiltration, and abnormal inherent properties of ossification of cartilage. Scherrer believed his case due to endocrine disturbance which may be added as another possible cause. They are usually discovered accidentally in the course of an examination of the ear. The affected portions of the ear is found to be of bony hardness. A roentgenogram will show the deposits of bone.

Small and sometimes large areas of the auricular cartilage may become ossified and can be felt on palpation.

The diagnosis is made by roentgenographic examination and biopsy taken from the ossified auricle.

### BENIGN NEOPLASMS OF THE EAR

#### Angioma

**Symptoms** — The bright red or lurid patches which are not elevated above the surface of the skin are not included in this group of tumors. The term 'angioma' as used here refers to the cavernous tumors which are bluish red in color and are made up of a series of venous sinuses or cavities of various sizes and shapes. They are often separated from each other by perforated fibrous septa which afford free intercommunication of their blood contents.

They may appear in the auricle in the canal or in both. They may be either primary or secondary extensions from adjacent structures. They vary in size but rarely grow larger than a small hen's egg. They are irregular in shape. Pulsation is occasionally present. Angioma is sometimes congenital while in other cases it develops after trauma or after the gradual dilatation of the blood vessels of the simple angioma. Cases are on record of angiomata which appeared after the auricle had been frozen.

The presence of pain depends chiefly upon the rapidity with which they grow. If of rapid development and large size the pain is consider-